DECEMBER, 1952

The Review of Gastroenterology

OFFICIAL PUBLICATION

NATIONAL GASTROENTEROLOGICAL ASSOCIATION

Choline Excretion Studies in Patients With and Without Diseases of the Liver

Clinical Pathological Conference

X-ray Therapy in Peptic Ulcer

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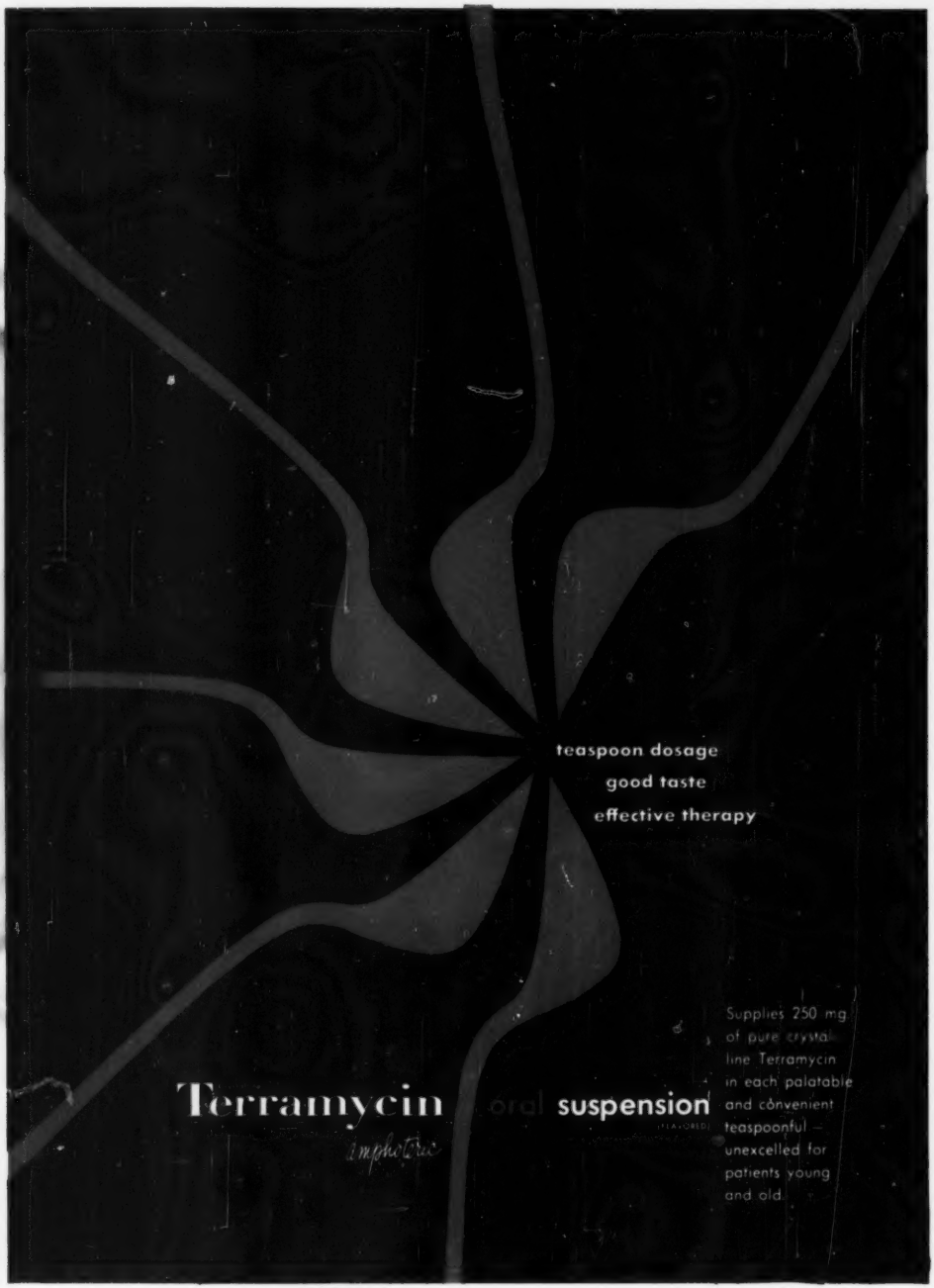
Eighteenth Annual Convention

Los Angeles, Calif., 12, 13, 14 October 1953

VOLUME 19 NUMBER 12



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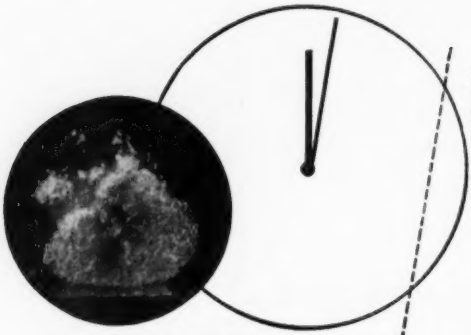

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(INCORPORATING THE AMERICAN JOURNAL OF GASTROENTEROLOGY)

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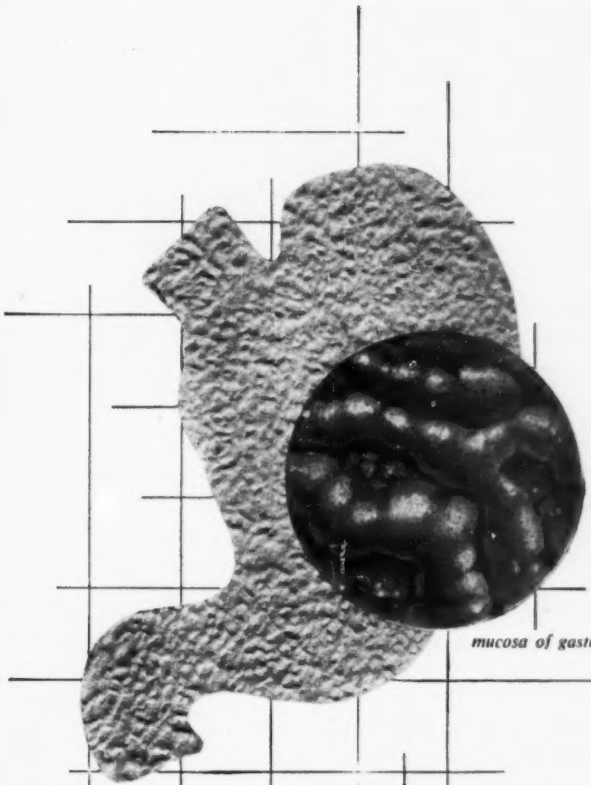

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1. Cass, L. J., and Wolf, L. P.: Gastroenterology *20*:149 (Jan.), 1952.
2. Berberian, D. A., Pauly, R. J., and Tainter, M. L.: Gastroenterology *20*:143 (Jan.), 1952.

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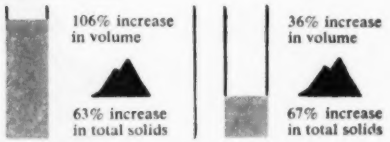

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CHOLINE EXCRETION STUDIES IN PATIENTS WITH AND WITHOUT DISEASES OF THE LIVER

Clinical Studies of Liver Dysfunction XVI

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Decatur, Ill.

In a previous study[1], we have shown that cirrhosis of the liver and carcinoma of the gastrointestinal tract have a similar defect in choline metabolism, in that a measurable excretion of choline occurred after a test dose was given to these patients. We would now like to record our results in liver disease in an attempt to determine the significance of choline excretion in these conditions. Very early in the present study, it seemed as though we had a new test for liver dysfunction. However, of all diseases of the liver, a measurable excretion by our test method occurs only in cirrhosis, and not at all in infectious hepatitis, infectious mononucleosis or homologous serum hepatitis. This may only indicate that the liver is able to carry out its many functions more satisfactorily in the latter three conditions than in cirrhosis of the liver.

Some recent work by Cayer and Cornatzer[2] casts some light on the meaning of choline excretion in cirrhosis. Using an isotopic technic they found that the administration of choline to animals on a choline deficient diet caused a marked stimulation of the turnover of liver and plasma phospholipids; this effect was only slight or not present in animals maintained on adequate diets. In patients with cirrhosis of the liver they found that an initial large dose of a lipotropic substance, e.g. choline, caused a marked increase in the rate of phospholipid

We are indebted to Dr. J. N. Cohen, Director of Medicine at the Greenpoint Hospital for the use of patients from the Medical Service; to Mr. F. J. Bandelin, Technical Director of the Flint, Eaton Co. for supplies and directions for the use of ammonium reineckate; to Dr. C. R. Kemp of Flint, Eaton Co. for advice and supplies of Syrup Chothyn.

turnover. This increase was no longer obtained after prolonged therapy. They concluded that the increase of phospholipid turnover reflects a condition of relative deficiency in lipotropic substances and that their test might provide an estimate of anticipated response to these substances.

Our work showed that when a large test dose of choline was given to patients with decompensated cirrhosis, a large excretion occurred. However, when these patients were treated satisfactorily with proper diet, vitamins and lipotropic substances, and were retested in one month to six weeks, they showed a much diminished or total lack of excretion. Those cases that did not improve continued to excrete large amounts of choline on retest (Table II). If we can equate increased phospholipid turnover with urinary excretion as in our study, we have a simplified test for anticipated response to lipotropic substances and evidence for lipotropic deficiency.

Since choline is utilized in the manufacture of phospholipid, it appears that larger amounts of choline are lost in the initial attempt at correction of the defect. The damaged liver cannot handle all the choline at first. As improvement occurs, the liver can more smoothly metabolize the choline, the rate of phospholipid formation is more uniform, and less choline is found in the urine. Our test, therefore, would most likely measure the same state of affairs, as that of Cayer and Cornatzer, and since it is extremely easy to perform could be used instead of the isotopic technic of these observers.

Our attention was first called to the possibility of using choline excretion as a test for liver function by an article by Castro-Mendoza, Jimenez Diaz and others[3]. These authors found that the urine of normal persons and of normal dogs either does not contain choline or contains it in very small quantities. They found that in normal persons and in normal dogs, a surcharge of choline chloride does not produce an elimination of the substance or else the elimination is increased in a very small measure. Experimental injury to the liver of dogs by the administration of carbon tetrachloride gave rise to a spontaneous cholinuria and to a marked cholinuria after surcharge. They stated that the same thing happens in patients who are suffering from liver disease (cirrhosis, hepatitis). They further stated that spontaneous cholinuria in patients with liver disease and marked cholinuria after choline surcharge in those patients, was due to poor utilization of choline by the liver, because of failure of the systems of fermentation, of the formation of phospholipids and of transmethylation.

In their tests they gave their patients 50 mg. per kilo of body weight of choline chloride over a 48 hour period, and tested quantitatively a 24 hour sample of urine for choline content. Our tests differed in the following particulars. We could not get a measurable excretion of choline in cases of cirrhosis with the recommended dose of choline chloride, even if given at one time. Therefore, we increased the dose of choline used for surcharge. We gave the patients 10 grams of choline as the dihydrogen citrate salt, since this was made available to us in

good supply. This is equivalent to 5 grams of choline as the chloride. Then to simplify the test, we tested semi-quantitatively (+ to ++++) the excretion of choline in three hourly interval specimens of urine up to the twelfth hour. Experience had shown that most of the choline was excreted in this interval if at all. While this maneuver simplified the test a great deal, it may have made it difficult for us to pick up early cases of liver damage.

TABLE I

DISTRIBUTION OF CASES STUDIED WITH CHOLINE EXCRETION TEST

Classification of Cases	Pos. Choline Test	Neg. Choline Test
Portal Cirrhosis	60	5
Infectious Hepatitis		15
Homologous Serum Hepatitis		8
Hepatitis (Inf. Mononucleosis)		6
Cholangiolitic Hepatitis		4
Control Patients (except as detailed below)	5 (slight)	68
Normal Controls (House staff)		15
Special Cases		
Carcinoma of gallbladder (Nodules in liver)		1
Congestive failure (chronic passive congestion)		16
Pernicious anemia		5
Common Duct Stone		12
Carcinoma of Pancreas (Liver metastases)	5	
Carcinoma of Pancreas (No obvious liver metastases)	3	
Carcinoma of Pancreas (Previously saturated with Choline)		1
Pancreatic Cyst	1	
Hodgkins' Disease (Enlarged Liver)	1	1
Hodgkins' Disease (Serum Hepatitis)		1
Hodgkins' Disease (Generalized)		2
Carcinoma of stomach (No metastases)	5	
Carcinoma of breast (Oseous metastases)	1	2
Gynecomastia (Cause undetermined)	1	
	82	162

The majority of patients with cirrhosis of the liver and with carcinoma of the gastrointestinal tract gave a positive excretion.

While we found that choline excretion was the rule after surcharge in cases of cirrhosis, we noted negative tests in cases of hepatitis (Table I). We found no excretion of choline after surcharge in normal people with several minor exceptions as will be detailed later. As we extended our study, we found that patients with carcinoma of the gastrointestinal tract also excreted large amounts of choline, when tested in the above manner. What this means in terms of mechanism will be discussed later.

Procedure

Ten grams of choline dihydrogen citrate was given as Syrup Chothyn (Flint, Eaton). Each dram contains 1 gram of choline as the dihydrogen citrate salt in a palatable vehicle.

A control specimen of urine was taken, as well as specimens at 3, 6, 9 and 12 hours. As stated above, this time interval was selected after tests showed that if choline was excreted at all, it was secreted during this interval.

To 10 c.c. of urine in a 15 c.c. centrifuge tube, add 1 c.c. of ammonium reineckate reagent.* Allow to stand for 30 minutes. A crystalline precipitate of lustrous pink plates indicates the presence of choline. This method indicates the presence of as little as ½ mg. of choline in 10 c.c. of urine.

We definitely identified the precipitate obtained in this manner from one of our cases of cirrhosis of the liver as choline reineckate.

We are aware of the fact that the chemical method measures only gross amounts of choline in the urine. A more accurate method is the biological determination, acetylating the choline and measuring the reaction on the isolated intestinal strip. An even more accurate determination is the microbiological method where choline is measured by the growth of a cholineless mutant of Neurospora crassa. These methods were beyond our facilities.

Unknown to us at the time that we conducted our tests, the pH of the urine is an important factor in the nature and amount of precipitate obtained. Thus it was shown that there are 4 different substances which may be present in urine capable of being precipitated by the reineckate method, and that precipitation in a slightly alkaline medium is necessary to insure complete precipitation of choline, without carrying down a considerable quantity of noncholine materials. We made no provision for different pH levels of the urine in our tests, so that if we were doing quantitative measurements, a very considerable error would be introduced.

According to an expert consulted by one of us[4], this limiting factor does not lessen the significance of our own findings, inasmuch as the noncholine materials precipitated according to our method may be just as important as the choline material.

In most of the subjects with liver disease the results of the choline excretion was correlated with bromsulfalein retention tests, cholesterol and ester determinations, albumin/globulin determinations, cephalin flocculation and the icterus index. Liver biopsies were done on twenty-five of the cases of cirrhosis and an attempt was made to correlate structure with function. The methods used for

*Ammonium reineckate reagent is prepared by dissolving 5 gm. of reinecke salt (Eastman Kodak) in 100 c.c. of absolute methyl alcohol. Proportionately smaller quantities may be used, and should be made up fresh every few days.

determining the liver functional tests were all standard procedures. The bromsulfalein tests were made by administering 5 mg. of bromsulfalein intravenously per kg. of body weight and taking a 45 minute blood specimen. Any retention of over 10 per cent in 45 minutes was considered abnormal. Cephalin flocculation tests were executed using Difco Cephalin and reading the result at end of 48 hours.

Results

Two hundred forty-four patients with and without diseases of the liver were tested by the choline excretion test as described above (Table I). With five exceptions, (60 of 65), patients with cirrhosis of the liver gave positive tests (i.e., excretion of choline in the urine after surcharge). There was a slight excretion of choline (+) at the twelfth hour in 5 aged chronic invalids. None of these patients gave evidence of liver disease by any of the usual liver function tests. Occasional abnormalities in liver function tests in normal aged individuals has been reported[5]. Thus, more than 90 per cent of the cases of cirrhosis of the liver excreted choline in the urine after surcharge. We feel that on the basis of a limited experience that the earlier the choline is excreted after surcharge, and the larger the amount of choline that is excreted, the worse the grade of cirrhosis.

The worst cases of cirrhosis of the liver had a large excretion of choline (+++ or ++++) after surcharge in the 3 or 6 hour samples. As the patient improved under therapy, which included the lipotropic substances, usually between 4 and 6 weeks, the choline excretion diminished in amount, and often became negative. Those cases that became worse clinically, continued to excrete choline after surcharge, no matter how long they were under therapy (Table II). Those cases of carcinoma of the gastrointestinal tract that excreted choline usually did so in the 9 and 12 hour samples. It cannot be stated definitely at this time whether the delay in excretion means a delayed absorption, as well as nonutilization, but this may well be the case.

Hepatitis patients did not give an excretion of choline after surcharge. It is on this basis that we feel that our test does not measure the finer grades of liver damage. It may also mean that a deficiency of lipotropic factors does not exist in hepatitis, and therefore, treatment with these substances would not be fruitful. This seems to be the prevailing opinion at present.

Discussion

Our work with choline excretion after surcharge shows that this "test" cannot be used as an indicator of hepatic disease, since it is negative in cases of hepatitis. It is true that it seems to mirror quite closely the condition of the patient in portal cirrhosis. Other than in cases of cirrhosis of the liver, we found that excretion of choline after surcharge occurred most often in patients having carcinoma of the gastrointestinal tract, with or without hepatic metastasis. Thus

TABLE II

PROFILES OF PATIENTS WITH LIVER DISEASE

Patient	History			Signs						
	Loss of Wt.	Alcoholism	Inadeq diet	Jaundice	Spider Nevi.	Coll. Circul. on Abd.	Splenomegaly	Hepatomegaly	Ascites	Avitaminosis
CJ–60	–	2 qts. beer qd. 15 yrs.	yes	marked	marked	present	–	3F	marked	+
IF–72	–	2 years occasional	–	slight	–	–	–	2F	–	+
AS–67	30 lbs.	beer & whiskey	yes	marked	4+	4+	–	2F	4+	1+
F deM–45	–	2 qts. beer qd. 15 yrs.	–	+	occ.	–	–	5F	–	–
JO–57	30 lbs. in 6 months	whiskey & beer 10 yrs.	yes	3+	present	–	1F	3F	–	+
FR–61	10 lbs.	occasional drinks	yes	3+	–	–	–	2F	–	+
NM–62	30 lbs.	drinks for 30 yrs. occ.	–	3+	–	3+	4F	10F	–	+
AC–62	–	beer & whiskey	yes	3+	+	+	1F	4F	3+	+
CS–56	30 lbs.	qt. whiskey qd. 15 yrs.	yes	3+	1+	–	1F	5F	–	marked
JM–47	10 lbs.	–	yes	1+	1+	–	–	3+	–	+
HR–53 (Hepatitis)	45 lbs. in 3 months	–	–	+	–	–	1F	2F	–	–

Profiles of Patients with Liver Disease.

The second choline tests in patients C.J. and A.S. were done 4 weeks after the first tests, and after therapy which included the lipotropic factors.

C.J. did poorly and died 6 months after the first test.

A.S. died 2 months after the first test.

Patient F. de M. clinically resembled a case of hepatitis although the biopsy revealed cirrhosis. His diet appeared adequate.

Patient J.O. did poorly and died about 9 months after the first test.

Patient F.R. improved markedly after lipotropic therapy.

Patient N.M. had massive liver enlargement, and although the liver biopsy revealed cir-

TABLE II

PROFILES OF PATIENTS WITH LIVER DISEASE

Laboratory Tests										
Ict. Index	Ceph. Floc.	Alk. Phosp.	Tot. Chol.	Chol. Esters	Sulpho-brom	Albu-min	Glo-bulin	Urine (Urobilinogen)	Biopsy	Choline Test: Cont. / 3 Hr. / 6 Hr. / 9 Hr. / 12 Hr.
12.2, 2.5	3+	3.9	199	35		2.9	3.6	neg.	Cirrhosis of the liver	– – 2+ 3+ 2+ – – 1+ 3+ 3+
7.5	2+	4.5			4% ret.	4.3	2.1	1+	Cloudy swelling, mild fatty changes	– – – 1+ 1+ – – – – –
11.1, 12.8	2+, 3+	16.8	156, 167	42, 67		3.9	1.7		Cirrhotic changes	– 1+ 2+ 4+ 4+ – 3+ 3+ 2+
26.7, 23.8, 30.3, 16.6	3+, 3+, 1+	4.2, 5.6	182, 140, 74, 222	46, 74, 143	40% ret.	3.5	3.1	2+, 2+	Cirrhosis	2 series neg.
10.4, 13.1, 1.9	1+, –, –, 2+	10.4, 11	233	156		4.0	1.7		Marked fatty changes	– – – 3+ 2+ – 3+ 2+ 1+ 1+
25, 60	–	9.9	115	40	32% ret.	3.7	1.9		Cloudy swelling, periportal fibrosis	– – 2+ 2+ 2+ – – – – –
23, 27, 35, 57, 27	3+, 1+	7.7, 8.6	322, 333, 392	122, 135, 156	24%	3.1, 2.9	3.6, 2.9	2+, 2+, 1+, 3+	2 specimens cirrhosis	2 series neg.
27	1+, 2+	18.5	175, 125	94, 66		2.5, 2.6	2.9, 2.5	4+, 2+, 4+	Cirrhotic changes	– +– +– 2+ 3+ – – – – –
13, 22, 23	4+, 2+, +–, 1+	5	125, 115, 161	53, 35, 50					Interstital fibrosis	– 1+ 1+ 2+ – – – – – –
10	1+					3.1	1.4		Fatty changes	– – – – –
28	4+, 4+, 4+	12	202	57%	100% ret.	1.9	4.9	15 ehrlich U.		– – – – –

rhosis, the negative choline tests may be due to the fact that the massive hypertrophy may have given this patient enough good liver cells to afford fair liver function. His diet apeared to be adequate.

Patients A.C. and C.S. did very well on lipotropic therapy and their choline tests done one month later were negative.

Patient C.S. also improved on lipotropic therapy.

Patient J.M. had pneumonoia and probably an associated hepatitis. His choline excretion test was negative. He had only a short history of alcoholism and inadequate diet.

Patient H.R. clinically resembled severe chronic hepatitis with posthepatic necrosis. He later developed ascites.

TABLE II (*continued*)

Patient	History			Signs						
	Loss of Wt.	Alcoholism	Inadeq diet	Jaundice	Spider Nevi.	Coll. Circul. on Abd.	Spleno-megaly	Hepato-megaly	Ascites	Avitam-iosis
LS–46 (Chorion cp. Hom. ser. Hep.)	–	–	–	1+	–	–	2F	2F	–	–
NM–31 (Tbc. peritonitis)	4 lbs. in 3 months	–	–	–	–	–	–	–	+	–
MM–58 (Cirrhosis)	–	–	yes	–	–	–	4F	3F	4+	+
WL–34 (Cirrhosis)	1 yr. unk. amt.	yes	yes	1+	–	–	–	2F	4+	+
CM–52 (Rt. Ht. D. with Ht. fail. Pseudochy-lous ascites)	2 yrs. unk. amt.	–	–	–	–	–	2F	3F	4+	–

Patient L.S. had homologous serum hepatitis.

Patient M.M. was thought to have cirrhosis on admission, but actually had tuberculous peritonitis proved by operation and biopsy.

Patient W.L. did very poorly and died about 1 year after the first test.

the choline excretion test cannot be used as a test for liver function or even for cirrhosis of the liver. However, if these results are confirmed, they may indicate that a similar pattern of liver dysfunction exists in cirrhosis of the liver and in gastrointestinal malignancy.

From our work in liver disease, we feel that the choline excretion "test" may help detect a condition of lipotropic deficiency and may also have some value in determining prognosis in cirrhosis patients. The greater majority of cirrhosis patients with liver failure excreted large amounts of choline after the test dose. Following adequate treatments for 1 month to 6 weeks, retest showed markedly diminished or absent excretion in those patients whose condition improved. Those patients, who after treatment did not show a markedly diminished or absent excretion on retest, died soon after.

Cayer and Cornatzer[2], using an isotopic technic, found that the administration of a large dose of choline to rats on a deficient diet, caused a marked stimulation of phospholipid turnover in the liver and plasma. This increased turnover was no longer obtained after an adequate diet was fed. The same situation was found to be present in patients with cirrhosis of the liver. The patients were given an intramuscular injection of radiophosphorus, and a large dose of lipo-

TABLE II (*continued*)

Laboratory Tests														
Ict. Index	Ceph. Floc.	Alk. Phosp.	Tot. Chol.	Chol. Esters	Sulpho-brom	Albu-min	Glo-bulin	Urine (Urobilinogen)	Biopsy	Choline Test Cont.	3 Hr.	6 Hr.	9 Hr.	12 Hr.
	4+	32	228			3.0	4.3			—	—	—	—	—
3.1	—		125	81%		2.1	3.0			—	—	—	—	—
12.2	4+		190	60%	100% ret.	1.8	4.5			—	—	—	2+	2+
										—	—	—	—	—
23.2	4+	9	135	55%		1.6	5.5	.25 ehrlich		—	4+	—	—	—
										—	4+	—	—	—
8	—		182	67%	100% ret.	2.7	2.5		Normal liver	—	—	—	—	—

Patient C.M. had 100 per cent retention of x bromsulfalein but negative choline tests. The marked dye retention was due to right heart failure and consequently poor transport of the dye to the liver.

tropic substance (choline or methionine) and the radioactivity of the lipoid phosphorus was determined. After several months of treatment the values were again determined. The amount of radioactive phosphorus in the plasma lipids was much higher in the early test than in the later tests. Although our measurements were not of this type, we also found consistent changes that could possibly be equated with Cayer and Cornatzer's results. In a severe case of cirrhosis that had not been treated, we found extensive choline excretion; after this same case was treated with lipotropic substances and diet, retest did not elicit choline excretion. Thus we might say that a high choline excretion on test probably reflected a state of relative deficiency of lipotropic substances and a need for this form of treatment.

This conclusion would be especially important in our work with carcinoma of the gastrointestinal tract. Others have also found evidences of hepatic dysfunction in patients with gastrointestinal carcinoma[6]. Our work is merely confirmatory; but it shows that the hepatic dysfunction is of a similar type as is found in cirrhosis of the liver. If excretion of choline in patients with gastrointestinal carcinoma means the same thing as it does in cirrhosis, it indicates a pressing need for lipotropic substances preoperatively and postoperatively. Patients with gastrointestinal carcinoma are subjected to extensive operations, with

prolonged anesthesia which are injurious to even normal livers[7]. These patients are also often given pentothal and other barbiturates which require detoxification by the liver.

Two additional burdens that they can ill afford to carry are sometimes placed on these patients. It has been shown by Ellinger[8] that x-ray induces fatty infiltration of the liver, which can be prevented if desoxycorticosterone is given. If the patient with carcinoma of the gastrointestinal tract already has a functionally insufficient liver, x-ray therapy could conceivably tip the balance.

Penicillin is used almost routinely during the surgical postoperative period. Therefore, it appeared to be of interest that the degradation product of penicillin in the body is penicillamine (beta beta dimethylcysteine). The structural formula of this compound resembles several of the essential amino acids, but if added to the diet of rats, it results in a severe deficiency syndrome, the symptoms of which include convulsions, running fits and finally death. Thus, it acts as an antimetabolitelike aminopiterin. Wilson and du Vigneaud[9] suggested that penicillamine acts by displacing one of the essential known amino acids, but neither cysteine, valine or threonine counteracted the ill-effects in the rats. Choline, however, overcame the deficiency, and amino ethanol was also effective.

Thus, load upon load is placed on a liver that is at best, functionally inadequate. We feel by virtue of our findings, and the others enumerated, that lipotropic therapy, especially choline, should be included in the preoperative and postoperative therapy of all patients with gastrointestinal carcinoma.

Summary

1. Evidence is adduced to show that a lipotropic deficiency exists in patients with cirrhosis of the liver and in carcinoma of the gastrointestinal tract. Profiles of patients with liver disease are included.

2. Other burdens on the liver are added to those already present in patients with carcinoma of the gastrointestinal tract by prolonged operations, anesthesia, x-ray therapy and penicillin treatment.

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CLINICAL PATHOLOGICAL CONFERENCE*

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Dr. Snapper:—We are going to take up the third case first. I will read the extract:

"This thirty-eight year old colored male first entered Cook County Hospital on February 10, 1949 with complaints of cough and intermittent pain in the right lower chest region of one month's duration. Physical examination revealed dullness and diminished breath sounds in the right lung base. A homogenous density of the right lower chest region was seen on the x-ray film. A thoracentesis was performed, and 60 c.c. of green, foul-smelling pus were removed."

No mention was made whether the heart was displaced.

In 1949 when antibiotics were being used, empyema of the pleura had already become extremely rare. The etiology of this pleural suppuration therefore merits close attention.

"The patient was treated with penicillin and sulfadiazine and was discharged after one month. Two months later he returned to the hospital complaining of chills, fever, night sweats and slight pain in the right side of the chest."

Chills, fever, and night sweats can be expected in empyema.

"There were dullness and absent breath sounds in the right lung base, and a repeat x-ray revealed a dense opacity occupying the lower fourth of the right chest. The right hemidiaphragm was elevated."

This is remarkable. Why should the right hemidiaphragm be elevated in an empyema?

"A thoracentesis yielded a thick, purulent material and a thoracostomy was eventually performed. A small subdiaphragmatic abscess cavity was discovered which yielded a moderate amount of purulent material."

Thus, in this case not only an empyema but also a subdiaphragmatic abscess existed.

"The patient was given penicillin and sulfadizine as well as emetine. The emetine was given empirically."

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This means that no amebae were found. In about fifty per cent of amebic liver abscesses, amebae are absent from the stool. Hardly ever does the first puncture of a liver abscess reveal the presence of amebae, because they live in the wall of the cavity. When on the next day, a second puncture is performed, then amebae are usually found.

"He was discharged after two months and followed in the chest clinic. For the next two months he continued to have a persistent cough with a blood-tinged sputum. He also complained of shortness of breath and was advised to re-enter the hospital and did so on August 24, 1949. Physical examination at this time revealed a poorly developed, poorly nourished colored, male, who was in no acute distress, but was moderately orthopneic. B.P. 116/78, p. 116, r. 32, t. 98.2°. Examination of the chest revealed the same abnormalities as those noted on his previous admission. The remaining physical examination was essentially negative. The patient was transferred to the Chest Service on August 26, 1949. One week later tube drainage was instituted for *emypema necessitatis*."

Apparently the empyema had perforated into the subcutaneous tissue.

"For several days thereafter a thick, purulent material drained. Chest x-ray and lipiodol instillation failed to show the complete extent of the sinus. The patient complained of liquid stools (evidently only in this late stage), shortness of breath, and severe right-sided chest pain. Further examination showed the liver to be six centimeters below the costal margin and four plus pitting edema of the lower extremities. Initial laboratory studies revealed: R.B.C., 3.20 million; W.B.C., 15,800; Hgb., 50 per cent; PMNs, 87 per cent; eosinophiles, 0; basophiles, 0; lymphocytes, 12 per cent; monocytes, 1 per cent."

Since the diaphragm was up and since the liver was down six centimeters below the costal margin, he really must have had a very large liver. The blood picture is compatible with the presence of pus.

"Urinalysis: Albumin, 1 plus; Sugar, 0; Microscopic negative, NPN 30 mg. per cent; Thymol Turbidity, 3.4 units; Cephalin Flocculation, 2 plus; gamma turbidity 8.2 units; Total Protein 7.7 gm. per cent; Inorganic Phosphate 3.7 mg. per cent; Alkaline Phosphatase 13.7 units; Icterus Index, 32."

Evidently obstructive jaundice with high icterus index and gamma turbidity due to hyperactivity of Kupffer cells.

"Stool specimen report was as follows: 'Endameba histolytica, macrophages, pus, large yeasts and occasional Blastocystis hominis were seen'."

As far as the finding of Endameba histolytica is concerned, the ameba were only seen. No cultures were made. I will, for the moment, disregard this positive report of the presence of Endameba histolytica, because I know by experience that these reports often have to be taken with a grain of salt.

"Chest films revealed rounded densities in the left costophrenic sinus and above the right diaphragmatic leaf. A right hilar mass was seen and there was

elevation of the right hemidiaphragm. EKG studies were normal. A gastrointestinal series revealed a normal upper and lower gastrointestinal tract. Treatment was chiefly symptomatic, consisting of: luminal grains 2, APC's, nembutal grains ¾, and codeine grains 2 daily. The patient was seen in medical consultation and placed on emetine grains 1 q. A.M., penicillin 300,000 units daily and streptomycin 0.5 grams q.i.d. He continued to have severe chest pain and hemoptysis. His hemoglobin dropped to 40 per cent. A subcutaneous swelling developed in the right chest region. This was aspirated and 200 c.c. of a foul, creamy pinkish material were obtained. Microscopic examination revealed many small round, oval refractile bodies which gave a positive stain with Sudan III. No growths were ever obtained on cultures of blood or pus."

The large liver of this patient does not function well, as follows from repeat laboratory studies made on October 15, 1949.

"Cephalin flocculation 3 plus, Thymol Turbidity 22.4 units, Gamma Turbidity 19.6 units, Alkaline Phosphatase 8.8 units, Cholesterol 127 mg. per cent, Icterus Index 20, NPN 20 mg. per cent, Total Protein, 7.4 gm. per cent. Albumin 3.1 gram per cent, Globulin, 4.3 gm. per cent. Urinalysis showed a four plus urobilinogen and no bile. The patient was reported coughing up a pinkish creamy pus similar to that aspirated from the subcutaneous abscess. A repeat chest film revealed diffuse mottled soft infiltrations in both lung fields and a homogenous density having a fluid air level in the right lower lung field. The temperature range was 99° to 101° during the hospital stay. On October 20, 1949, a thoracostomy was performed. Two draining sinuses connecting the skin with a honeycombed pleural space were discovered. The diaphragm was elevated but aspiration in all fields failed to yield pus. The anterior empyema cavity was digitally explored, but no deep communication was determined. The patient apparently survived the procedure in fair condition."

The patient after operation is always "in fair condition".

"Five hundred c.c. of whole blood were given postoperatively. In spite of the above measures, the patient continued a downhill course and expired on October 21, 1949, approximately nine months after the onset of his illness."

We have here a patient who has not only an empyema of the pleura but also pus between the diaphragm and the liver. I would say that hardly any diseases proceeds from the pleura to the subdiaphragmatic space and the liver. Therefore, in this case the original disease must have been below the diaphragm.

This patient has a tremendous liver. There are several liver diseases which perforate through the diaphragm and bronchi and thereby cause secondary lung disease. This holds true for hydatid cysts of the liver and for amebic liver abscesses.

It is said that Endameba histolytica has been found in the stool. Can we accept that this patient suffered from a tropical liver abscess, perforating through

the diaphragm and giving rise to empyema and lung lesions? I would say no, because this patient received emetine and under this treatment a liver abscess which has been drained should have healed.

In view of the efficacy of emetine treatment, I would be willing to exclude a tropical liver abscess. In addition, there was a subcutaneous accumulation of pus in the right chest. This should never have happened in an amebic liver abscess, drained and treated with emetine.

Finally, during the thoracostomy, two draining sinuses connecting the skin with a "honeycombed" pleural space were discovered. Why should amebiasis give rise to "honeycombing" of the pleural cavity? It might give rise to granulomas or hemoptysis but not to a honeycombed pleural space. We, therefore, have to consider a disease which comes from below, which perforates through the diaphragm, gives rise to empyema, hemoptysis and sinus formation, and to a honeycombed pleural cavity. This course of events indicates the presence of a fungus disease.

As far as I know, blastomycosis is very rare in Chicago, but actinomycosis is frequent.

The latter disease could well cause a subphrenic abscess, an empyema, which does not heal after drainage or antibiotics, with subcutaneous sinuses and a honeycombed pleura. Where is the primary focus? Primary actinomycosis of the liver is rare. As far as I can remember, I have seen only one such case.

Maybe the diarrhea indicates that there was intestinal actinomycosis. Since there was a right-sided empyema, the right lobe of the liver was probably more involved than the left one. Diseases of the ascending and transverse colon affect the right liver lobe; disease of the descending colon the left liver lobe. Thus the diagnosis of actinomycosis with a possible primary localzation in the ileocecal area seems to be in order.

Dr. Wangensteen:—This is a very interesting patient and I found that Dr. Snapper's dissection of the problem followed closely my own. I would go along with him in the thought that this is primarily, in the beginning, a subdiaphragmatic lesion.

Dr. Howard Beye, of Iowa, reported a study of cases of empyema at the University of Iowa Surgical Clinic about 30 years ago. He said, as I recall it, that in a large number of empyemas he had only once seen extension of the disease from the pleural cavity into the peritoneal cavity; whereas the reverse is the rule; that is, a subdiaphragmatic infection frequently leads to infection of the pleura. This sequence helps very much in combined abdominal and thoracic infections to trace the origin of the trouble.

Pathologists occasionally speak like Delphic Oracles. In a Clinical Pathological Conference like this, at our own University Hospital a number of years

ago, on the basis of Beye's observations, I was able to persuade our pathologists to change their diagnosis from a conjectured extension of an infection from the pleura to the abdomen via the blood stream. It concerned a man with pancreatic calculi, a peripancreatic collection of exudate and an empyema. When the sequence of peripancreatic suppuration and extension through the diaphragm was suggested, our pathologists graciously changed their diagnosis.

What are the possibilities in the case now under discussion? I think the first one we should review as plausible, but perhaps not the most likely, is staphylococcal or pneumococcal infection. There is no history of skin infection, such as carbuncle or of furuncle of the kidney, going into the perirenal fat. Perforation of the diaphragm, empyema—lung abscess—that is a possibility, but certainly at some time or other, the staphylococcus or pneumococcus should have been found in the exudate.

Spontaneous perforations from the umbilicus, low hemoglobin, and all the general picture of pleural infection can certainly come from that agency, and I don't think we should dismiss it altogether, but I would merely write it down as a possibility.

Now, I am not a student of amebic dysentery. I know very little about it. It would have been nice to have had here a statement about the stool. We do know the patient had diarrhea. Did he have an ulcerative colitis? There is no mention of a proctoscopic examination. We do not know whether he had ulceration in the bowel, as would be suggested by the presence of blood and pus in the stool. Secondly, we cannot exclude the possibility of amebic abscess, in the light of the data put before us. And, as Dr. Snapper said, secondary pyogenic infection of an untapped amebic abscess is practically nonexistent.

Leonard Rogers, in the British Foreign Medical Service, was knighted for his work in indicating that it was perfectly safe to tap an amebic abscess with an aspirating needle, because of the absence of secondary pyogenic invasion. I have seen amebic abscesses which have been drained. There is one statement that usually goes along with amebic abscess pus: it is a sort of mucoid exudate—which apparently is not the situation here.

So, I agree with Dr. Snapper, this could not possibly be accounted for on the basis of amebic dysentery alone.

Now, are there any other possibilities? Could it be tuberculosis? Yes, it could possibly be turberculosis, but why should a tuberculous abscess perforate from beneath the diaphragm? It would have had to have some origin there. Could this be tuberculosis in the pleural cavity and amebic dysentery beneath the diaphragm? That is a possibility—but not a very likely one.

I should have liked very much to have had the opportunity of looking at the x-rays myself to see what these shadows are like. I have seen the spontaneous dissection of pus in tuberculosis in the manner that was described in the protocol.

In my experience these are usually large circular shadows in the pleura with dissection abscesses in the anterior chest wall—the so-called *empyema necessitatis.*

Is the situation explicable on the basis of one disease alone? If it were, I would agree with Dr. Snapper that, the disease would be actinomycosis; however, there is one thing absent. We should have a statement suggesting that x-ray studies indicated thickening of the ribs as well as the presence of intrapleural shadows. I have seen actinomycosis several times originate in the area around the liver and go right through the liver, then through the diaphragm, and give a pyopneumothorax. However, here it was said that there were changes in both

Fig. 1—Liver abscess connected with empyema cavity which, in turn, communicates with pulmonary abscesses.

thoraces—on both sides, as I remember it, a shadow—in the left thorax as well as in the right thorax. Is that correct?

Dr. Popper:—Mostly right.

Dr. Wangensteen:—Wasn't there a shadow on the left?

Dr. Popper:—Some fluid.

Dr. Wangensteen:—I have not seen a patient with actinomycosis who also had the disease on the other side.

We used to think with German observers, led by Israel, who first described the organism, actinomyces in pus, that it was essentially of bovine origin; that you got actinomycosis in the following way: you were associated with cattle, that the cows had actinomycosis, lumpy jaw, and went out and ate grass; and the farmer came along and chewed the grass which had actinomyces on the blades of grass.

Frederick Lord, of Boston, used to insist that actinomycosis was an endogenous disease and later, Emmons, of the Public Health Service, in Puerto Rico, demonstrated to everyone's satisfaction that actinomycosis is primarily an endogenous disease. You and I might have it. A couple of bacteriologists from Chicago said we carry about 57 varieties of organisms in our mouths, and I think actinomycosis is as frequent in New York City as on the farms in Minnesota.

Naeslund, in Copenhagen, has shown we have these organisms in salivary ducts around the teeth and under the proper provocation (which we do not know) these invade the tissues about the jaws and neck. You never see any focus when you cure the patient or get the mouth open. And, as Dr. Snapper said, if this is actinomycosis, it might have come from the cecum, with an abscess in the liver which burrowed right through the liver. If it is actinomycosis, there will probably be some actinomycotic lesions in there, but the very circumstance that there were lesions on the left side of the thorax too makes me feel a bit uneasy over making a diagnosis of actinomycosis. If it is, it is in my experience very extraordinary, so I think the diagnostic possibilities come down to two, in the end. One is tuberculosis plus amebic infection of the liver—not a very satisfactory suggestion. The other is actinomycosis alone. There is much to substantiate this suggestion, and only one thing against it — the bilaterality of the thoracic findings. The weight of the submitted evidence favors an extensive actinomycosis of abdominal origin.

Dr. Hans Popper:—At autopsy a pleural empyema was found on the right side communicating with the thoracotomy wound as well as with several pulmonary abscesses in the right lower lobe which apparently have broken into the pleural space. Some of these abscesses partly drained by large bronchi coalesced to a cavity of apple size revealing a shaggy wall. In addition, many more lung abscesses up to cherry size were found, some of them also in the left lung. They were filled with a fibrino-purulent exudate and lined by a granulation tissue rich in segmented leucocytes, histiocytes (many of them foam cells) and some giant cells; in places it underwent fibrosis. In the surrounding lung tissue thrombosed vessels and acute and chronic bronchi-pneumonic foci were noted. The tracheobronchial lymph nodes showed only little reactive hyperplasia. The diaphragm lining the empyema cavity revealed fibrotic thickening. Its pleural surface was covered by a thick yellow shaggy membrane. Below it a liver abscess about 12 cm. in diameter was located in the upper portion of the right hepatic lobe. Over its dome the diaphragm and the adherent hepatic capsule

were thinned out in an area about 6 cm. in diameter in the center of which the abscess communicated with the empyema cavity (Fig 1). The liver abscess itself was lined by a similar shaggy yellow membrane containing segmented leucocytes, histiocytes and many fat-containing foam cells, characteristic of chronic infection and responsible for the grossly visible yellow color. There were several more smaller partly communicating hepatic abscesses with scalloped walls and a dry necrotic content. Microscopically, the wall of several of them contained granules composed of branching gram-positive filaments and on the periphery of eosiniphilic clubs, characteristic for Actinomyces bovis (Fig. 2). Around the abscesses scarring and collapse of the liver tissue was noted but otherwise no inflammatory or degenerative changes were seen in the liver tissue; the proliferation of the Kupffer cells found is probably responsible for the serum gamma globulin elevation and the abnormal flocculation tests. The spleen was large and revealed inflammatory reticuloendothelial hyperplasia. The kidneys showed interstitial

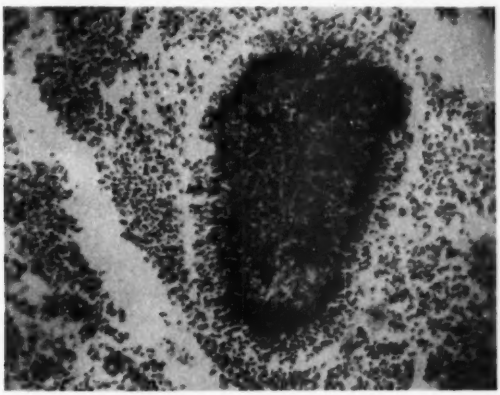

Fig. 2—Gram stain, 130 X. Actinomycosis granule in liver abscess. Radiating filaments with peripheral clubs.

inflammatory infiltration with a few giant cells and endophlebitis, the latter apparently on an allergic basis. The intestine, including the appendix, was free and no amebic lesions were found.

If we try to summarize the findings in this case, we want first to remember that actinomycosis, as a rule, starts either in the jaw, lung or appendix and extends from there without any respect for organ borders, quite often producing fistulae. We are safe to assume that in this instance the process started in the lung, approximately nine months ago. At that time chest pain on the right side, cough, fever and chills developed. Six months later anemia and leucocytosis is recorded. The extension to the liver probably took place two months before death with resulting hepatomegaly and probably compression of the vena cava and some elevation of icterus index and alkaline phosphatase. Finally, and

probably only recently, the actinomycosis spread by hematogenous and bronchogenic routes in the lung and a superimposed nonspecific infection was grafted upon it accompanied by high fever and expectoration of pus in the preterminal period. The renal involvement may also be either a hematogenous spread of the actinomycosis or a nonspecific allergic reaction. I would thus assume that the actinomycotic process started in the lung and extended downwards into the liver in contrast to the usual route of amebiasis.

Dr. Wangensteen:—We will now take up the second case. "In September 1949, one week before admission to Cook County Hospital, this 47-year-old white male electrician experienced mild abdominal pain with nausea. This was followed by a bowel movement of bright red blood; subsequent bowel movements were tarry and continued to be so for three days. In 1921 he had had gonorrhea but denied having had syphilis. Otherwise he had been well except for twenty years previous to admission when he had some occasional gas pains which were diagnosed as appendicitis; and for the last ten or fifteen years he has had some gas pains intermittently. Seven years ago he disclosed that he had consumed large amounts of alcohol until a few years prior to his admission. At that time he had been treated for substernal pain and pain in the lower posterior thorax."

That might be significant.

"The evening before admission the patient developed abdominal cramps and 'gas pains' shortly after eating some hot tamales. He took some effervescent salts, and the pain continued for an hour and a half. He slept well that night and felt no discomfort upon arising next morning. He had a cup of black coffee for breakfast and about thirty minutes later he became nauseated and vomited black, coffee-like liquid."

It would be very nice to know if he vomited blood. It is hard to know from that statement.

"The vomiting was followed by abdominal cramps and a bowel movement of congealed, dark red blood. At this time he experienced also some blurring of vision.

"On physical examination the temperature was 99°, pulse 96/min., and respiration 20/min. Blood pressure was 110/64. He did not appear ill but he was said to be described as 'ill at ease' and 'nervous'. The temporal arteries were palpable. There was some displacement of the cardiac apical impulse to the left. The prostate was moderately enlarged, firm and smooth; the stool was burgundy red in color. Neurologic examination was negative. Blood examination showed an RBC of 3.25 cu. mm., hemoglobin 50 per cent, WBC 7,000/cu. mm. and a differential of polys, 73 per cent, lymphs. 17 per cent, and monos. 10 per cent. Urine had a specific gravity of 1.022, was acid in reaction, and otherwise negative.

"He was sedated with morphine and atropine, and given Tinc. Belladonna. The remainder of treatment consisted of a bland diet, hourly milk and cream

feedings, and calcium carbonate and magnesium oxide powders. Blood transfusion was ordered as indicated by the blood count. X-ray, P-A, of the chest, on the second hospital day, was negative.

"Early on the third hospital day, he again had a tarry stool. His pulse was thready, B.P. 90/60, and he was perspiring. Red blood cell count at this time was 1.75 mill./cu. mm. His nail beds were pale, but his general color was good. Abdominal examination was negative and bowel sounds normal. After blood transfusion, his red cell count was 2.49 mill./cu. mm. Later the same evening he complained of intermittent cramps and lower abdominal pain. He was sweating and restless with a B.P. of 100/46. Again he had a bloody stool.

"He still had cramps on the fourth hospital day, with continuing bloody stools. In the afternoon he was out of bed and complained of severe abdominal pain and fell to the floor. The pulse and blood pressure were unobtainable, and he was pronounced dead a few minutes later."

Well, I suppose the dissection of this problem relates wholly to decision as to where in the gastrointestinal canal the bleeding originated, and on the basis of the evidence given, it would be very difficult to say. If that early statement relating to coffee-grounds vomiting has any meaning, it would suggest a duodenal ulcer crater with a blow-out of the gastroduodenal artery and death from hemorrhage.

There is no suggestion in the history or physical examination of a palpable mass in the abdomen. The sudden death would suggest the blow-out of a vessel larger than the gastroduodenal—such as rupture of an aneurysm. But how did the blood get into the gut or the stomach? I am not aware that a dissecting aneurysm will bleed into the gut. However, it is repeatedly stated in the record that the patient had gas pains; that he had red blood in the stool, a circumstance which suggests bleeding low down in the tract. The repeated gas pains suggest the presence of some mildly obstructive mechanism in the bowel.

I think the differentiation comes down to the manner in which we analyze these two circumstances. It would be easy to conclude on the one hand that he had a terrific hemorrhage from an eroding duodenal ulcer crater, or the terminal event might have been a cardiac one, because one of the fairly common denominators in patients with peptic ulcer is coronary sclerosis. This is one solution which I think would be completely satisfying were it not for the circumstance that we are told the blood in the stool was definitely red.

What sort of lesion in the bowel could give this sequence of events? Well, a chronic intussusception could do it; yet the hemorrhagic features are much too pronounced. Could he have a tumor in the bowel, of some sort, such as a leiomyoma with ulceration, or a hemangioma?

I confess that I find it very difficult to resolve this situation in a satisfying manner. I would like to know more about the nature and source of the hemorrhage than is available in the data given us.

A diagnosis of bleeding duodenal ulcer is not an acceptable diagnosis in the presence of red blood in the stool. What large vessel ruptured which was giving red blood in the stool and intermittent gas pains? I must confess that I hesitate to make a diagnosis but I would favor rupture of a very large vascular hemangiomatous tumor of the gut which periodically caused intermittent obstruction.

Dr. Snapper:—This man had hematemesis and melena at the same time. He, therefore, must have had a terrific hemorrhage which must have originated in a very large blood vessel. The severity of the bouts of hemorrhages is further illustrated by the fact that he ultimately died suddenly from an acute bleeding. Very few patients with duodenal ulcers die of hemorrhages unless they are transfused too heavily. Patients who died from intestinal hemorrhages usually suffered from a liver cirrhosis. In this case report no enlargement of the spleen is mentioned. The hemorrhage of patients with a peptic ulcer or liver cirrhosis is hardly ever so excessive that they die so suddenly as this patient did. Such a sudden death due to hemmorhage indicates rupture of an artery, not of a venous varicosity as is the case in liver cirrhosis.

In this patient, perforation of a large artery, probably of an aneurysm of the aorta, seems possible. Even the fatal hemorrhage due to erosion of the pancreaticoduodenal artery by a duodenal ulcer is hardly ever severe enough to cause a sudden death as happened to this patient.

Periarteritis nodosa which also causes ruptures of large arteries is improbable in view of absence of a shift to the left, high blood pressure, splenomegaly, neuropathy, etc.

My experience is that a terrific intestinal hemorrhage leading to sudden death is usually due to perforation of an abdominal aneurysm of the aorta.

Often a flat plate of the abdomen is of help in these cases, but even in the absence of x-ray data this diagnosis has to be considered.

Dr. Hans Popper:—At the autopsy the brain was edematous and showed some small encephalomacic foci as well as arteriosclerosis of the cerebral vessels. The kidneys were pale and anemic. Histologically they showed brown pigmentation due to hemosiderin probably as a result of hemolysis. Stomach and duodenum contained a large blood clot forming a cast of the dilated lumen. A similar cast filled the jejunum. In the search for the site of the gastrointestinal hemorrhage an ulcer was noted, almost 1 cm. in diameter, with ragged edge and base in the esophagus just above the cardia. On histologic study it was a squamous carcinoma which could have been the cause of the hemorrhage. However, on following the gastrointestinal tract further down, another ulcer, again about 1 cm. in diameter, was found in the posterior wall of the duodenum a few centimeters below the papilla of Vater (Fig. 3). Its edges were only slightly elevated and not injected. The localization of this ulcer is unusual and speaks against a simple peptic etiology since below the papilla of Vater the neutralizing

effect of the pancreatic juice should have made itself felt. Under the microscope its edges revealed the beginning of fibrotic separation and fraying of the muscle fibres suggesting that the ulcer should be some weeks old. In other places frank ischemic necrosis of the tissue implicated a vascular occlusion. The explanation of the dramatic gastrointestinal hemorrhage was a communication of the base of the ulcer with an aneurysm of the abdominal aorta of walnut size which developed just below the origin of the inferior mesenteric artery (Fig. 4). The thoracic aorta was free but the abdominal aorta exhibited severe arteriosclerosis suggesting, on first glance, an arteriosclerotic etiology of the aneurysm, as it is typical for this location. Histologically, however, marked necrosis was seen in the fibrotic wall of the aneurysm although the reticulum fibres were preserved in the necrotic part. Obliterating phlebitis was also noted and plasma cells were found around thick-walled arteries. Moreover, in a lymph node adjacent to the aneurysms there were large areas of necrosis (Fig 5). All these findings strongly suggest a syphilitic origin of the process.

We, therefore, have demonstrated an unusual location of a syphilitic aortic aneurysm associated with a gummatous change gradually eroding the wall of

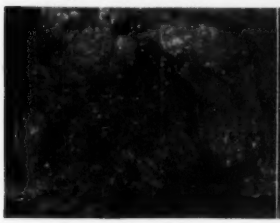

Fig. 3

Fig. 4

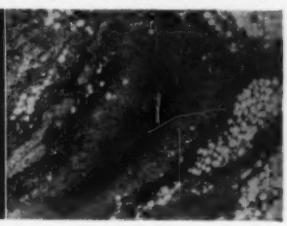

Fig. 5

Fig. 3—Recent ulcer in duodenum below the papilla of Vater.
Fig. 4—Syphilitic aneurysm of abdominal aorta breaking into duodenum.
Fig. 5—H & E stain, 82 X. Gummatous changes and endo- and periphlebitis around syphilitic aneurysm of abdominal aorta.

the duodenum and finally perforating into its lower portion. Syphilitic aneurysm in this location with perforation into the gastrointestinal tract has, to our knowledge, only once been reported in the literature.

The liver showed central necrosis due to anemia and the pancreas revealed some early interstitial infiltration.

The clinical pathologic correlation is rather simple. We do not know how long the patient had syphilis. Negative serologic tests are found in a fair percentage of patients with syphilitic aortitis. This condition probably explains the abdominal pain going back for seven years. The aneurysm associated with the gumma produced the acute duodenal ulcer which, only ten days before death, led to the clinical symptoms of abdominal pain, nausea and melena. The gradual perforation is reflected in the intermediate cramps and finally led to acute anemia and shock.

Dr. Snapper:—"The third case is a 15-year old colored female who first entered Cook County Hospital on April 12, 1946 at the age of eleven years. She stated that except for measles and whooping cough in childhood she had been well until March, 1946, when migratory upper abdominal pains developed. These were followed in a few days by headache, malaise, anorexia, nausea, and 'yellowing' of her eyes. She lost about five pounds in weight. Physical examination essentially normal, except for the presence of icteric sclerae, a firm, smooth, slightly tender liver that was enlarged five fingers breadths below the left costal margin. The urine gave a positive test for bilirubin, but a negative reaction for urobilinogen and sugar. The Kahn test was negative. The hemoglobin was sixty per cent and red count 3.75 million with no sickling on a wet preparation. The white count was 4,200 with 65 per cent neutrophiles, 9 per cent eosinophiles, and 22 per cent lymphocytes. The erythrocytic sedimentation rate was 38 mm. in one hour. A sternal marrow examination revealed a shift to the left in granulopoiesis and an increase in eosinophiles. The icterus index was 36 units, total protein 6.6 grams per cent, with 4.0 gram per cent globulin."

This patient did not suffer from an uncomplicated hepatitis. If during the course of a hepatitis, the serum globulin increases, patients always develop liver cirrhosis. This patient has a large spleen and a high globulin—she must have a liver cirrhosis.

"Total serum cholesterol 240 mg. per cent with 160 mg. per cent as esters is a normal ratio. Alkaline phosphatase, 32 units, much increased."

This patient has a cirrhosis but at the same time signs of regurgitation of bile are present as indicated by increase of the alkaline phosphatase.

"The cephalin flocculation test was 2 plus, the fecal urobilinogen on two occasions was low, 66 and 12 mg. per 100 grams of stool. The urine urobilinogen was normal, 2.3 and 2.2 mg. per two-hour specimen."

Dr. Popper:—In jaundice, low.

Dr. Snapper:—"The nonprotein nitrogen was 30 mg. per cent. During this three and one half month's stay in the hospital she did not respond to penicillin, vitamins or emetine treatment."

Emetine treatment is superfluous in this case because in tropical liver abscess jaundice never occurs.

"She ran a febrile course. Her temperature spiked frequntly as high as 104° and was associated with chills."

That is not too rare in hepatitis and in certain kinds of liver cirrhosis.

"Blood cultures were negative. Anterior cervical lymph nodes became palpable bilaterally. The first heterophile agglutination test done on May 2, 1946 was positive in a dilution of 1:640 and a month later was positive in a 1:80 dilution."

In so-called glandular fever or mononucleosis, jaundice may appear. I have never seen cirrhosis follow the jaundice in mononucleosis, but the possibility has to be considered.

"No pathological lymphocytes were noted on the blood smears."

That makes mononucleosis extremely improbable.

"Agglutination tests for typhoid and brucella were negative. The icterus index had risen to 80 units and the alkaline phosphatase had increased to 90 units at the time the patient signed her release."

Both the presence of liver cirrhosis and obstructive jaundice are certain.

"Throughout the next three years she remained jaundiced and entered the hospital on four occasions for study. A low grade fever with temperature up to 102° and chills were present during each admission. She lost weight progressively. Clubbing of the fingers developed."

This is sometimes seen in liver cirrhosis.

"The liver remained enlarged and the spleen became enormous, extending into the pelvis. The superficial veins of the abdomen became distended."

This means obstruction of portal circulation.

"Only occasionally did the urine show a positive test for urobilinogen, however, bilirubin was always present. The liver profiles changed only slightly from those of her first admission. The globulin returned to normal and the serum cholesterol rose to as high as 420 mg. per cent. There were 55 per cent cholesterol esters—a low normal value. Her final admission eleven days before death was on September 9, 1949, (which is three and a half years after the first admission) because of abdominal swelling for two months. She was severely emaciated and markedly jaundiced. There was pitting edema over the ankles and sacrum. The fingers were clubbed. There was shifting dullness in the abdomen and after a paracentesis of six liters of a greenish brown, turbid fluid, the firm liver edge was palpated four fingers breadth below the right costal margin and the spleen was noted to occupy the entire left abdomen. Spider angiomata were present on the abdomen. The red blood count was 3.74 million, the white blood count was 6,200 with 84 per cent neutrophiles . . . "

Too many neutrophiles!

". . . seventeen of which were bands . . ."

Much too many!

". . . and 14 per cent lymphocytes. The total protein was 4.4 grams per cent with 2.1 grams per cent globulin."

The total protein was low now, which explains the edema, but the globulin was no more increased.

"The total serum cholesterol was 85 mg. per cent, 39 esters – both values being very low. The alkaline phosphatase was 4.9 units, the icteric index 96 units, the cephalin cholesterol flocculation 0, thymol turbidity 6.8 units–that is, increased–and the nonprotein nitrogen was 38 mg. per cent, a normal value. Her hospital course was rapidly downhill. The ascites returned, coma developed, and death ensued on September 20, 1949, four years after the onset of her illness."

She must have had a liver cirrhosis–in view of the large liver, large spleen, jaundice with signs of liver insufficiency. At one time during the disease the cholesterol was very high. The latter sign often leads to the erroneous diagnosis of xantomatous liver cirrhosis. It should be emphasized that in initial stages of cholangiolitic liver cirrhosis, where chronic inflammation around the smallest bile ducts ultimately leads to liver cirrhosis, the serum cholesterol is always very high. Only in the later stages of the disease, the cholesterol decreases to very low values. A gradual rise of the serum cholesterol, decreasing rapidly in the last months before death, is one of the characteristic signs of cholangiolitic liver cirrhosis. In view of the presence of the combination of liver cirrhosis, and the signs of regurgitation of bile and high alkaline phosphatase, in association with the initial hypercholesterolemia, I would favor the diagnosis of cholangiolitic liver cirrhosis.

Even when the main part of the liver shows the signs of cholangiolitic liver cirrhosis, here and there small areas of Laennec's liver cirrhosis may be present.

Fever occurs often in cholangiolitic hepatitis and cholangiolitic liver cirrhosis. I am, however, somewhat troubled by the result of the paracentesis, consisting of six liters of a greenish-brown, turbid fluid. In a liver cirrhosis with hypoproteinemia and ascites, the color of the ascites fluid should not be green and turbid, but yellow and clear. Therefore, a complication must be present. One could speculate that a hepatoma could have developed on top of the liver cirrhosis, which would modify the qualities of the fluid. However, a hepatoma usually causes a loud, arterial bruit over the liver which is not mentioned in the protocol. One can only speculate about the complication. The diagnosis, cholangiolitic liver cirrhosis, seems, however, certain.

Dr. Hans Popper:–We have from this patient two biopsies, one three and and one two and one-half years before death. In the older specimen (Fig. 6) we noted some nodules suggesting cirrhosis formation already at that time. In addition, small granulomatous accumulations of histiocytes, lymphocytes, plasma cells and only few segmented leucocytes were noted in the portal triads and around the intralobular cholangioles. Bile duct proliferation was marked, but relatively little liver cell damage was seen. The second liver biopsy specimen showed hardly any cirrhosis formation which clearly indicates the spotty character of this process, easily missed in a needle biopsy specimen. Otherwise, the picture was similar suggesting the diagnosis of chronic cholangiolitic hepatitis.

At the autopsy about 2,000 c.c. of an amber fluid were found in the peritoneal cavity. The liver was slightly enlarged for a girl of this age, weighing 1,400 gm. On the surface fibrinous exudate was noted as an expression of a chronic peritoneal irritation. On the green-brown cut surface some connective tissue increase, but not a frank cirrhotic picture, was seen. The extrahepatic bile ducts were free and not dilated. Microscopically (Fig. 7) the lobular pattern was preserved, excluding a Laennec's cirrhosis; however, fibrosis was noted throughout in the periportal areas, around the central vein and between the liver cells separating especially on the lobular periphery individual cells. The bile duct proliferation in the portal triads, so impressive in the biopsy specimen, had disappeared; as a matter of fact, in many triads the bile ducts were missing. Also the inflammation appeared burned out and only a few plasma cells persisted. This burning out of the inflammatory reaction is in keeping with the change of the originally

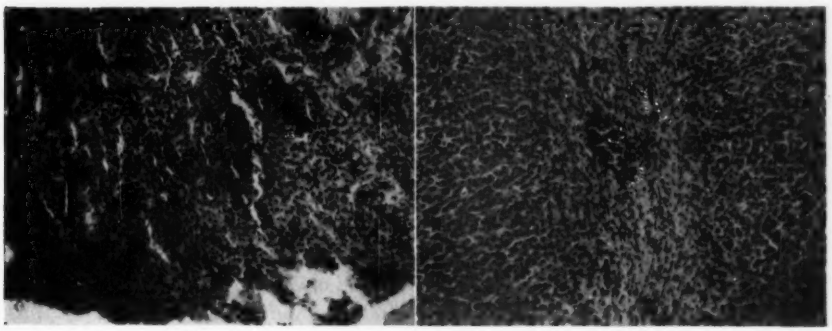

Fig. 6 Fig. 7

Fig. 6—Liver biopsy specimen of cholangiolitic hepatitis. H & E stain, 130 X. Florid inflammatory infiltration in portal triad and around intralobular cholangioles. The portal triad reveal some bile duct proliferation.

Fig. 7—Liver at autopsy revealing cholangiolitic cirrhosis. H & E stain, 100 X. Fibrosis of enlarged portal triad which reveals scanty inflammatory infiltration and hardly any bile ducts. Interstitial fibrosis separates the liver cells at the periphery of the lobule. The arrangement of the liver cell plates is intact.

positive flocculation tests to the norm. We assume that elevation of the alkaline phosphatase is related to the cholangioles; during their proliferation the enzyme activity was high, to drop when the cholangioles became choked by the inflammation. The total cholesterol, as an expression of persisting biliary obstruction, rose to very high levels. The portal intralobular connective tissue proliferation resulted from fibroplasia and not from mere collapse of the framework. The picture is thus that of a cholangiolitic cirrhosis of Hanot type, possibly the end result of a viral hepatitis, though some additional features complicated it. For instance, in some places connective tissue trabeculae coalesced to form septa which distorted the lobular pattern and this is probably responsible for the portal hypertension and ascites which are not characteristic for cholangiolitic cirrhosis. Furthermore, the intralobular fibrosis compressed the liver cells and

this, together with the intrahepatic biliary obstruction, caused hepatocellular necrosis leading to hepatic coma.

The spleen, 1,200 gm. in weight, was very much enlarged and revealed marked fibrosis in the pulp as well as reticulum cell hyperplasia. The splenic vein was normal. Fibrocongestion cannot be the entire explanation of the splenomegaly since the portal hypertension was probably recent; it may, therefore, be justified to put the splenic fibrosis in analogy with the hepatic fibrosis. There was a chronic interstitial pancreatitis. Dr. Snapper considered as the "red herring" of the story the turbidity and high protein content of the ascitic fluid. It was explained by an encapsulated intraperitoneal abscess near the transverse colon which was probably produced by the paracentesis. The glomeruli of the large kidney revealed marked fibrotic thickening of the basement membrane resembling somewhat the "wire loop" changes of *lupus erythematosus;* there was also some interstitial fibrosis and in the medulla bilirubin casts were seen. Over the heart, which was of normal size, an abacterial fibrinous pericarditis was noted. There was also fibrinous material over the pleura. The alveolar septa of the lung were thickened due to fibrosis. The periaortic lymph nodes were large and revealed a remarkable degree of reticuloendothelial and especially lymphoid hyperplasia which produced some superficial resemblance to infectious mononucleosis. The marked brain edema probably was the final cause of death.

Trying to correlate the anatomical with the clinical findings, we have to assume that this girl had a clinically silent acute hepatitis. We consider a viral etiology in the absence of any clues for any other etiologic factor. The marked lymphoid reaction in the lymph nodes would also support a viral etiology. The slight resemblance to infectious mononucleosis becomes apparent in lymph nodes in chronic viral hepatitis pointing to the close relation of viral hepatitis and infectious mononucleosis. Four years before death the girl was seen with jaundice, anorexia, malaise, splenomegaly, leukopenia and spiking fever and at that time a florid cholangiolitic hepatitis with transition into cirrhosis was present, which was associated with intrahepatic biliary obstruction and its laboratory manifestation. At that time a very active fibroplasia took place in liver and spleen. We feel that the stimulus for the connective tissue cells to form fibres also causes the reticuloendothelial cells to pour gamma globulin into the blood reflected in hyperglobulinemia and abnormal flocculation tests. This could also apply to the connective tissue diseases. Anyway, in this case some resemblance to these diseases may be noted in the fibrosis involving several parenchymal organs. The florid process burned out and was replaced by scarring, which in the liver caused portal hypertension and ascites. The unusual degree of splenomegaly and also the generalized fibrosis may be related to the young age. Finally, the continuous fibrotic compression and biliary obstruction make themselves felt upon the liver cells which fail (reflected in the drop of total and ester cholesterol) and thus usher in the fatal brain edema.

X-RAY THERAPY IN PEPTIC ULCER*

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The capricious disease of peptic ulcer has its cause hidden in mystery, its course is variable and much of its therapy is based on assumption and some even on no logic whatsoever.

Statistics of cure of peptic ulcer are almost worthless. That is because ulcers are of varieties of pathology and size, vary in location, and patients' age and sex must be considered. To state definite figures takes advantage of credulity and of the lack of experience of the medical man and patient alike. Yet, with all this, I am making bold to present an old method, which after forty years, has been rediscovered and which in my opinion has always had a special value in the medical handling of peptic ulcer when no complications exist.

Physical and mental rest, nonirritating diets, antacid remedies, and acid-controlling surgical operations bring benefits by the control of gastric acidity. In the absence of complications and taken in large groups, the cases in which the acidity is controlled fare better than those in which this had not been accomplished. Surgery accomplishes its benefits by control of acidity; medicine undertakes to accomplish the same. It is possible that the use of banthine bromide for the control of vagal conditions will prove to be a valuable addition to the medical handling of peptic ulcer, more so than the use of belladonna or atropine. While the effect of banthine and belladonna (atropine) in the control of the vagotonic state (which accompanies most cases of peptic ulcer) is desirable, acidity control in the direct way is also desirable (alkalis, milk, surgery). It is not possible as yet to state whether the continued use of banthine would produce as good a result in the cure of an ulcer as could the x-ray, and whether perhaps both should be used in the medical handling together with diet, antacids, etc. While foods in the stomach stimulate the secretion of gastric juice, the neurological, emotional and personality factors that operate through the parasympathetic are likewise important. There is only meager and insufficient data so far on the effect of banthine on the secretion of gastric acidity.

It was observed in the early days of x-ray therapy that one could depend upon a reduction of gastric secretion in the majority of cases after a dozen or more deep x-ray treatments. In those days hyperacidity, the symptom of hypersecretion was associated mostly with dietetic indiscretion, so-called acid gastritis in the acute cases, and with the then so-called Reichmann's disease in the continued cases. The significance of excess secretion in ulcer, especially duodenal, was not known then, and there is no doubt that many of the cases of so-called Reichmann's disease were instances of peptic ulcer, mostly duodenal. Some years

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after the teaching of the Mayos that duodenal ulcer was a chronic disease requiring surgery, the writer reported in a group of one hundred cases that approximately one-third were curable by medical means. In all of these x-ray therapy was employed. There is no doubt in my mind today that the use of the x-ray both in curability and control of recurrences is the best medical treatment we have and often will take the place of surgery. Attention is drawn to spontaneous healing, to psychotherapy, and the dietetic and antacid treatments in the results obtained. In one-third of the cases of gastric ulcer treated by x-ray, recurrence of symptoms occurred, contrasting with about 80 per cent in cases treated medically without radiation. In cases in which achlorhydria was definitely reduced for the time being, only 17 per cent had recurrence, and in no case with persistent achlorhydria did the ulcer recur.

One must keep in mind that while an acute ulcer can heal in a few days, the majority are chronic in nature. Various efforts have been made to get some idea under ideal conditions of how long it would take for an ulcer to heal by the usual methods of treatment. There is a belief that this takes from four to six weeks, but manifestly this would vary in different cases as they are seen clinically.

Ricketts, Kirsner, Humphrey's and Palmer[1] present that moderate irradiation produces an acute inflammation of the gastric wall, characterized gastroscopically by hyperemia, hemorrhage, edema and adherent exudate, and histologically by degenerative changes in the epithelial cells and lymph follicles, increased cellular infiltration and relatively minor changes in blood vessels. Atrophy of the gastric mucosa often ensues which is uniformly present in patients with postradiation anacidity. It is the writer's opinion that the x-rays have a special effect on the glandular cells, both central and parietal.

The x-rays have a depressing effect on all the gastric secretions, postprandial, night or fasting. An occasional case will not show any reduction of acid, but these do not represent ten per cent of all instances. The why of this is not understandable. It possibly is due to the parietal cells in some individuals not being sensitive to the atonizing effects of the rays, or incomplete dosage for that individual. The duration of depression produced by radiation is variable. Levin, Hamann and Palmer[2] report that in 14 of 15 cases the depressing effects of the x-ray was still present after the crater of the ulcer could no longer be demonstrated radiologically. In the majority of the cases, however, the reduction can be depended upon to persist for months, long enough to accomplish a worthwhile result in the healing of the ulcer.

The first mention of the therapeutic use of the x-ray occurred in 1909[3] and was repeated in the first edition of my text book[4]. With bismuth subcarbonate in the stomach, exposures of small doses of x-rays were made over the entire organ. Predicated upon the beneficial use of the x-rays in skin conditions, etc., it was proven that in a considerable number of cases the excess secretion of HCl became lessened and occasionally absent. With varying success the simple

method of one to three dozen treatments over the stomach area was carried out for a number of years. How much actual value was accomplished by this means alone was not possible to evaluate with any degree of accuracy. A large number of cases were treated and about ten years ago with the improvement in apparatus the method was more standardized.

It has been known for years that the proper use of the x-rays to the stomach produced a depression in gastric secretion. This had been attested to by Bruegel[5], Bensaude[6], Emery[7], Palmer and Templeton[8] and others. According to Ricketts, Palmer, Kirsner and Hamann[9] histamine tested cases of depression of gastric acidity in over all results in 423 cases was considerably dependent on the number of roentgen units employed. The results ranged from a 9 per cent achlorhydria in 666-1000 r to a 76 per cent achlorhydria when 1600-2500 r were employed. Practically all showed a reduction. The reduction occurred proving that the effects of the x-ray was on the secreting cells. The duration of the reduction is variable in different instances lasting from less than 30 days to several years. In about two-thirds of the cases of gastric ulcer the achlorhydria lasted less than one month, the rest lasting much longer. In duodenal ulcer this was less marked. In these cases of duodenal ulcer satisfactory results were obtained in 352 cases out of more than 700 (50 per cent) and in 50 cases of gastric ulcer healing occurred in 90 per cent. When one considers that in duodenal ulcers by medical management alone only about one-third are cured and that this is doubled, and that 60 per cent of gastric ulcer is raised to 90 per cent, it is obvious that distinct benefits can come from the proper use of the x-rays. The lesser per cent in duodenal ulcer as compared to gastric ulcer is due to the higher incidence and persistence of acid secretion in the duodenal ulcer. Obviously only by reduction of the acid or total achlorhydria could one hope for results, and this is not uniformly accomplished. Nor is it possible to foretell in other than the achlorhydic instances whether healing would be brought about. Another factor to contend with is the possibility of duodenal ulcers penetrating and these modify the results that may be accomplished. In some cases a permanent achylia is produced perhaps due to an atrophic gastritis, which produces no symptoms. In 9 cases out of 50 with gastric ulcer, the hydrochloric acid returned to previous levels, and the aforementioned authors report that this occurred in two-thirds of those who received less than 1100 r. Irradiation achlorhydria does not produce symptoms, but in the beginning of the x-ray treatments a few days mild nausea and even slight increase of ulcer pain may be experienced. The disappearance of the niche and healed result as proved by x-ray films and gastroscope cannot be taken as positive signs of healing to the extent that one can depend upon long continued absence of clinical symptoms. Deformity of the duodenum may persist, the ulcer apparently being permanently healed.

Only the body and fundic end of the stomach is treated because these are safe from the ulcerating effects of the x-ray as observed in the antrum. The treatment may be carried out with patients of any age or sex, and duration

of symptoms. Because where achlorhydria has been established and lasts for three months the ulcers usually healed completely, full doses of x-ray should be given in all instances. It is true that tissue susceptibility varies but there is no way to judge what is the sufficient x-ray amount to produce an achlorhydria. No harm has come with doses of 2000 r in 10 to 14 days time and Brick has shown that no harm occurs in double and triple this dose. In the report of Ricketts, Palmer, Kirsner and Hamann 1600 to 2500 r significantly reduced the acid in more than 50 per cent of all patients with gastric ulcer, and 83 per cent of those with duodenal, and the lesser doses were effective in only 23 per cent.

There is a majority of cases in which continued anacidity is not produced, but in the majority it is definitely depressed. The depression may not occur until several weeks after the x-ray treatments are discontinued. If a markedly lowered acidity is produced no further testing is necessary since it can be depended upon that totally or in part it will last long enough to carry on an "ulcer cure". If it is not distinctly lowered it is well to estimate the acidity again, say in two or three months after the cessation of the x-ray treatments. There is then no objection to conducting another course of treatments because in some people resistance to the effects of the x-rays is very high. Nor is there any objection to a second course of treatment especially if recurrence takes place, when almost invariably it will be noted that the acid is high again. As to further x-ray procedures one must use judgment. Instances of pyloric obstruction of the edematous type (not the organic) and hemorrhage are suitable for x-ray treatments. In the last three years there were eleven instances of definite pyloric obstruction (which is very liable to come on or be made worse by a recurrence) of the nonorganic type. In each of these but one the ulcer was healed and the pyloric obstruction disappeared. In the one case the obstruction proved to be of the organic type secondary to duodenal ulcer, and while the case became achlorhydic, operation was called for because of pain from penetration of the ulcer into the pancreas. There were twenty-six cases in which frank hemorrhage was present or had been in the past. The occult bleeding cases are not included. Of these, 21 were duodenal and 5 gastric and in these, 17 became symptom-free. All had cessation or marked reduction of acid. In four the acid reduction was only slight or too temporary, two had hemorrhages, one in 6 and the other in 8 months after treatment, and they both were operated upon. These four cases were duodenal. According to Ricketts, et al[1] recurrences in gastric ulcer seemed to have been reduced from 80 per cent to one-third of the patients, and in duodenal ulcer from two-thirds to about one-third in those who received the larger doses. The absence of recurrence despite recurrence of acid, which returns to previous secretory levels, occurred in approximately two-thirds of the patients. Of 120 cases in 1945 and 1946 both duodenal and gastric, recurrences occurred in 46, a little less than one-half. These developed in the usual times from 6 months to 3 years, all were distinctly acid at the time of the recurrence and 11 were again x-rayed with apparently four good results.

For the treatment of ulcer it is desirable to immediately obtain the index of acidity. This being high, a bland diet, phosphagel, and banthine are prescribed together with additions of physical and mental rest and advice on personality and emotional matters. X-ray treatment is started about a week after the initiation of medical treatment and the acid is again estimated three or four weeks after the termination of the x-ray treatment. According to the status of the acidity the medical treatments are modified. In anacidity the antacid treatments are discontinued, the diet being continued. It is well not to temporize with a gastric ulcer too long. Absence of healing or early resumption of symptoms should suggest surgery since about 6 per cent or more of all gastric ulcers are malignant. The cases should be examined at intervals for a year or two even when the results seem to be good.

The technic followed in cases in the past has been variable. Up to about 1920 no apparatus could be depended upon to deliver definite amounts of x-ray, the depth factor, the proper use of filters and means to measure the x-rays. A 12 unit coil with a "hard tube" and oiled leather filter were employed with exposure over the whole stomach, the exposures being three in a week and stopped at skin erythema. During the 1930's and 1940's the technic was somewhat better standardized but even here it was variable and it was not as controlled as in the last ten years.

The method employed the last three years is essentially that reported by Ricketts, Palmer, Kirsner and Hamann[8] with some slight modification as suggested by Dr. Adolf Abraham. The majority of cases being duodenal ulcers larger doses were employed throughout all of the cases.

The treatment consists of taking a film of the stomach, especially the fundic end, using bismuth instead of barium. Predicated on this film, the location of the fundic end and body of the stomach is marked on the surface of the body, in front and in back. Treatment consists of 200 kilovolts, one copper and one Al filter being used. Two hundred roentgen units are given, one each day, one day in front and one day in the back, to the fundus and body of the stomach, not the pylorus. The area covered is about 15 cm. x 15 cm. and 10 cm. x 10 cm. for a small person. Ten to 12 treatments are used according to the thickness of the body.

Conclusions

The x-ray therapy practically doubles the medical curability of peptic ulcer, and no doubt will save cases from surgery.

The results in gastric ulcer are somewhat better than in duodenal ulcer.

The x-ray work should be properly done and there is no danger if this is so.

The presence of nonorganic pyloric obstruction or frank hemorrhages would not interfere with the results possible to be obtained.

In the presence of uncomplicated peptic ulcer with definite gastric acidities the therapeutic use of the x-ray is worthy of a trial. This may be repeated in the instance of recurrence where the acid had been established to original amounts.

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THE SURGICAL ANATOMY OF GROIN HERNIA

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Compton, Calif.

Plastic operations for the repair of herniae in the groin are associated with a minimum recurrence rate if pertinent anatomic structures are recognized and correctly utilized at the time of surgical reconstruction.

Embryonic development of the testes, particularly in relation to peritoneal investments is an important factor in the formation of inguinal herniae, and serves as a logical introduction to the subject.

The testes are formed high in the lumbar region, between the peritoneum and the transversalis fascia. They descend and about the third month of intrauterine life occupy a position in the iliac fossa close to the pelvic brim. Motive power for this migration is furnished by hormonal influences from the anterior lobe of the pituitary gland and by smooth muscle fibres, the gubernaculum testes connecting the lower pole of the testicle with the corium of the scrotal sac. Contemporarily from the pelvic peritoneum, at the level of what is to be the internal inguinal ring, two buds are formed which elongate distally beneath the arching fibres of the internal oblique muscle and assuming a sac-like shape, pass through the subcutaneous inguinal ring into the scrotum. This processus vaginalis peritonei which communicates proximally with the peritoneal cavity forms an incomplete double sheath about the testes and spermatic cord. The upper portion of this sheath surrounding the vas and accompanying structures, is the funicular process, the lower portion covering the testicle and epididymis becomes the tunica vaginalis testes. During the course of normal maturation there is a fibrous obliteration of the sac from the internal inguinal ring to the superior pole of the testis resulting in a solid cord, the obliterated funicular process. The tunica vaginalis testes, however, maintains a patent cavity between it's visceral (testicular) and parietal (scrotal) layers, the locale for hydrocele formation.

Several abnormal variations of development arrest may occur facilitating the formation of herniae or the accumulation of serous fluid along the course of the processus vaginalis (Fig. 1).

1. Complete patency of the processus vaginalis from the internal inguinal ring to the lower pole of the testis. This situation precedes the formation of congenital inguinal hernia which is always of the indirect type.

2. Obliteration of the processus vaginalis at the superior pole of the testis with complete patency of the funicular process up to, and including the internal inguinal ring. This is associated with an indirect type of congenital hernia into the open funicular process, i.e. a funicular hernia.

3. Segmental obliteration of the processus vaginalis at (a) the internal inguinal ring and at, (b) the superior pole of the testis, leaving a patent funicular process between these two points. This condition is usually accompanied by a collection of fluid within the unobliterated portion of the sac, forming a hydrocele of the cord. An acquired indirect hernia existing in conjunction with such a hydrocele produces an encysted inguinal hernia.

Anatomy of the Inguinal Canal

The inguinal canal may be regarded as a potential intermuscular and interfascial channel which has been weakened in the male by the descent of the testes and by variations in the normal obliteration of the processus vaginalis peritonei. In the female, the inguinal canal is occupied by a solid structure, the ligamentum teres uteri, and the peritoneal sheath surrounding it proximally, (the homologue of the vaginal process in the male) is normally obliterated in late intrauterine life. Distaff inguinal herniae are therefore uncommon. When they do occur, the

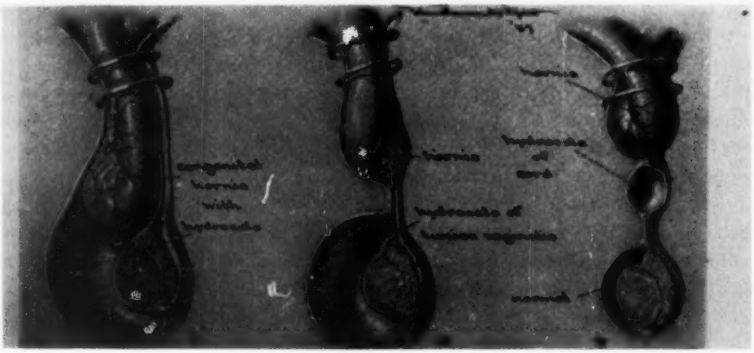

Fig. 1

peritoneal investment of the round ligament remains patent, forming the canal of Nuck. Because of ovarian and Fallopian tube proximity to the abdominal inguinal ring, these structures are frequently found in the hernial sac.

Assumption of the erect position with it's tendency to approximate the superficial and deep inguinal rings in the same sagittal plane also predisposes to hernia formation.

The inguinal canal is about two inches in length and extends obliquely from the abdominal (internal) inguinal ring, downward and medially to the subcutaneous (external) ring. The latter is easily felt in the male as an orifice of varying size in the external oblique fascia, just lateral and superior to the pubic spine. In the female, due to the fanlike insertion of the round ligament it is very small and barely palpable. The layers of the abdominal wall covering the canal

are: skin; superficial (Camper) fascia; subcutaneous fat; deep (Scarpa) fascia; external oblique aponeurosis and arching muscular fibres of the internal oblique covering the canal laterally.

Certain portions of the internal oblique and of the obliquus abdominis transversus deserve special identification and attention. These take origin from the lateral third of the inguinal (Poupart) ligament, and pass medially to form the fibromuscular structure known as the falx aponeurotica inguinalis (conjoined tendon). The falx forms the roof and medially in combination with the transversalis fascia, part of the posterior wall of the inguinal canal (Fig. 2).

The deep epigastric artery, a branch of the external iliac arising just proximal to the inguinal ligament is an important structure differentiating direct and in-

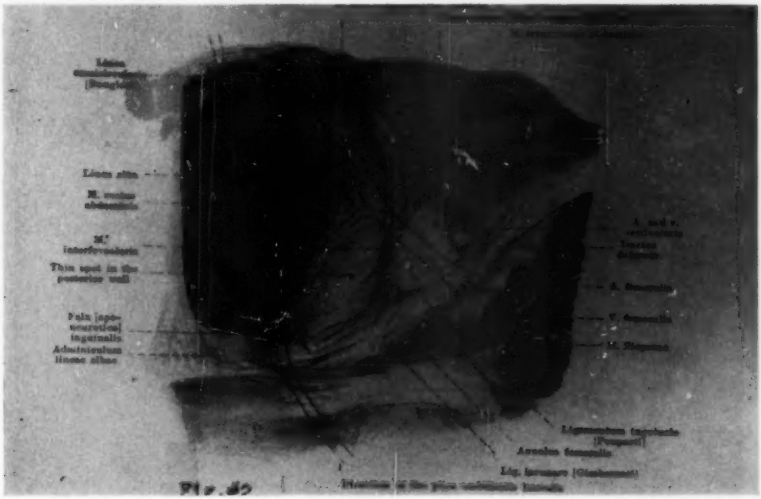

Fig. 2

direct inguinal herniae at operation. This vessel passes upward and medially at right angles to the long axis of the inguinal canal between the peritoneum and the transversalis fascia. The abdominal inguinal ring is situated lateral to the artery, hence a direct inguinal hernia originates medial to this vessel, whereas an indirect hernia enters its peritoneal sac lateral to it. During any dissection in this region, the inferior epigastric vessels should be carefully exposed and their relation to the internal hernial orifice determined.

The inguinal (Poupart) ligament is the free, inferior edge of the external oblique muscle. It is entirely fascial in structure and extends from the anterior superior iliac spine to the pubic tubercle. After inserting firmly on the tubercle it sends a triangular band of fascia laterally along the inner portion of the ascend-

ing pubic ramus forming the lacunar (Gimbernat) ligament. The lacunar ligament extends along the pubic ramus to the medial edge of the fossa ovalis thus forming one of the boundaries of a femoral hernia. In 1938, Anson and McVay published the first of their anatomic studies which relegated the inguinal ligament and the falx aponeurotica to positions of minor importance in hernia repair. Substituted for the falx was the obliquus abdominis transversus together with its underlying transversalis fascia. The place of the inguinal ligament was taken by a structure whose identity and relationships require more precise description. Anson and McVay describe a ligamentum pubicum superius (Cooper) as forming a substantial fascial layer along the pectineal line of the ascending pubic ramus. There is reason to agree with the accuracy of this study. Several pioneer anatomists, among them Spalteholz, designate this superior pubic ligament as being confined principally to the symphysis pubis and having the function of

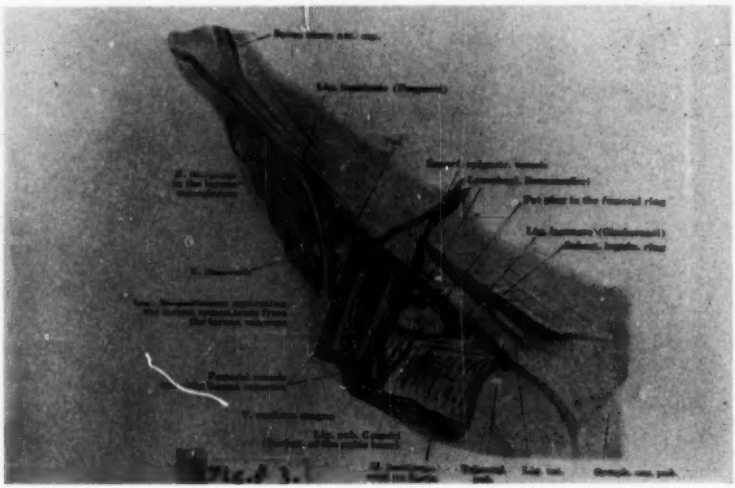

Fig. 3

maintaining apposition of the two pubic bones. The ligament consists of strong decussating fibres connecting the pubic spines with each other and with the tendinous insertion of the rectus abdominis. Laterally this ligament is prolonged along the pectineal line and forms an origin for the pectineus muscle. It is the pectineal extention of the pubic ligament which is of interest in this revised concept of hernial repair. One anatomist stated that any cadaver can be easily suspended by a strong tape passed under its pubic ligament, so great is its supporting potential (Figs. 3 and 4).

If the researches of Anson and McVay are accepted, one structural defect can be blamed for the occurrence of all groin herniae, whether indirect, direct, or femoral. This defect is a thinning of the posterior inguinal wall produced spe-

cifically by an attenuation of the obliquus abdominis transversus and of the transversalis fascia.

Anatomic Repair of Inguinal Hernia

For many years surgeons had been reasonably satisfied with some variations of the original Bassini reconstruction. The essential features of these operations were: (1) high ligation of a cleanly dissected sac; (2) approximation of the fascial portion of the falx aponeurotica inguinalis to the so-called "shelving edge" of the inguinal ligament. Whether to complete this approximation above or beneath the spermatic cord; whether to leave the cord subcutaneous as in the Halstead repair and what the choice of suture material should be, were un-

Fig. 4

settled questions, which each surgeon answered to his own satisfaction as personal experiences increased.

If it is agreed that posterior inguinal wall defects in the transversalis fascia are responsible for hernia formation, then logical method of surgical repair might proceed as follows: (1) Skin incision parallel to the inguinal ligament and slightly above it from the abdominal inguinal ring to the pubic tubercle. The abdominal ring is projected on the skin at a point midway between the anterior superior iliac spine and the pubic tubercle. Anson and his associates use a transverse incision in this region postulating that it follows more closely Langer's stress lines in the skin and gives a better cosmetic result. The incision in either case is deepened to expose the external oblique aponeurosis. Three substantial

blood vessels are consistently found between Camper's and Scarpa's fascia superficial to this aponeurosis; (a) superficial circumflex iliac, (b) superficial epigastric, (c) external pudendal and they should be isolated, clamped, cut and tied affording a bloodless dissection. Fine cotton (#100) or silk seems preferable for this purpose. (2) The subcutaneous inguinal ring is next identified and the external oblique fascia split parallel to its fibres starting midway between the superior and inferior crura of the ring. This is easily and best accomplished by a gentle pushing motion of the half-opened scissors. The supero-medial flap is then separated bluntly from the underlying muscular belly of the internal oblique until its fusion with the anterior rectus sheath is reached. This fusion line is in a sagittal plane almost two inches medial to the semilunar (Spigeli) line marking the outer edge of the rectus abdominis. This point is important and justifies

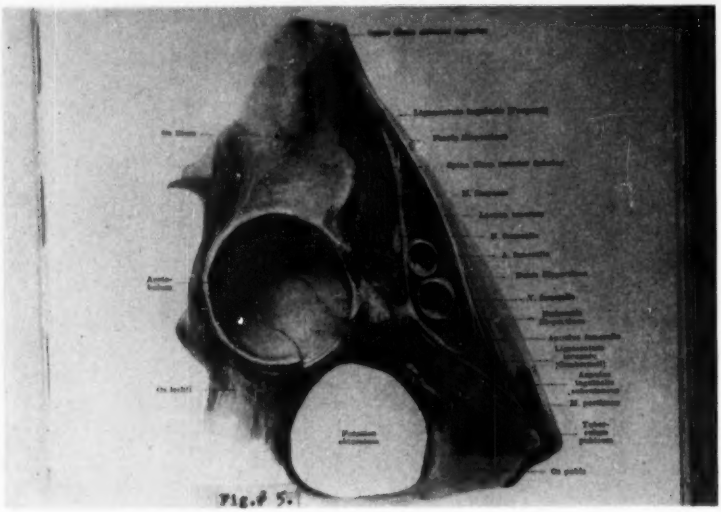

Fig. 5

the relaxing incisions advocated by McVay which otherwise would further weaken the floor of Hesselbach's triangle if the external oblique insertion into the rectus sheath were situated more laterally.

(3) The infero-lateral flap is similarly separated until its free lower margin, the inguinal ligament, is exposed. The spermatic cord covered by attenuated layers of fascia will now be plainly in view. These fascias are derived from the layers of the anterior abdominal wall through which the testes pass to the scrotum. They are, (a) internal spermatic fascia (transversalis); (b) cremasteric fascia (internal oblique); (c) external spermatic fascia (external oblique). After these layers are removed by sharp and gauze dissection to the internal inguinal ring, the cord is examined for the presence of an indirect hernial sac. This, if

present, will be found most readily in the region of the internal ring, and is antero-medial to the cord structures, being the only reduplicated fold of serous membrane attached to the funiculus. The sac is opened and its communication with the peritoneal cavity verified. With the cord and the internal oblique retracted gently upward, the floor of the inguinal canal is exposed. This consists of obliquus abdominis transversus, transversalis fascia and peritoneum. Any weakness in this layer associated with a direct hernia will manifest itself as a definite bulge intensified, if regional anesthesia is being employed, by having the patient cough. Its firmness can be further checked by passing one finger through the abdominal inguinal ring and palpating the area from within. In the presence of a double or pantaloon hernia, consisting of both a direct and an indirect sac, Houget's maneuver is valuable. This consists of exerting traction on the medial wall of the indirect sac, pulling the redundant tissue of the direct hernia lateral to the inferior epigastric vessels, thus converting the situation into peritoneal closure of a single larger indirect sac. A hernia of the direct variety, when existing alone, usually occurs in one of two clinical forms: (1) a large diffuse protusion involving practically all of Hesselbach's triangle with no tendency of the sac to become pedunculated or, (2) a smaller localized defect with a tube-like process of peritoneum which may elongate sufficiently to pass into the scrotum through the superficial inguinal ring. This type refutes older concepts that direct inguinal herniae never enters the scrotal sac. Disposition of the sac varies in these two types. Where the hernial orifice is small and the sac pedunculated, it may be conveniently treated by dissection, high ligation, and excision. When repairing a diffuse extensive defect, opening the sac and attempting excision of the redundant peritoneum serves no useful purpose. A better procedure is to carefully split the transversalis fascia over the protrusion, tighten the parietal peritoneum with imbricating mattress sutures, and form a new inguinal floor by any logical method of fascial repair. This fascial reconstruction is mandatory in every direct hernia regardless of size and in all large indirect herniae which by medial extension weaken Hesselbach's triangle. It is also the surgeon's duty to meticulously inspect the entire groin region for defects elsewhere, even after one definite sac has ben found.

The type of repair is the moot question. The prevailing method has been, and possibly still is, an approximation of the falx inguinalis and the inguinal ligament. In the hands of surgeons who have judiciously isolated and disposed of the hernial sac and who have sutured the falx without undue tension to the inguinal ligament, this operation probably gave satisfactory results in the majority of patients. There are present, however, major theoretical objections to this technic which will surely manifest themselves to the thoughtful, critical surgeon.

First is the inadvisability of utilizing a structure as relatively free as the inguinal ligament in an attempt to create a firm supporting floor. Even worse is the possibility, in the case of a large direct hernia, of having this protrusion slip beneath the ligament and appear in the femoral region. That such a recurrence

does take place, is evidenced by reliable statistics in large series of hernioplasties. Lastly there is the frequent difficulty of bringing fascia from the falx down along the entire extent of inguinal canal. It is necessary to approximate tissues of similar structure, fascia to fascia if a substantial and lasting repair is to be expected. Medially the falx is invariably tendinous but as it passes laterally to become the inferior edge of the internal oblique, the transition to pure muscle fibre occurs. The temptation to suture the inguinal ligament to this structure is frequently too great to be resisted, jeopardizing the effectiveness of such a reconstruction.

On practical as well as conjectural grounds, McVay's utilization of the pectineal fascia would appear to overcome the above objections. It is a firm, unyielding structure having the additional advantage of being placed inferior to the femoral vessels rather than superior to them as is the inguinal ligament (Fig. 5). This anatomic feature serves to close the femoral fossa to abnormal protrusions from Hesselbach's triangle when Cooper's ligament, from the symphysis pubis to the medial side of the femoral vein is approximated to the transversalis fascia above the direct hernial orifice. When this type of repair is performed, a longitudinal relaxing incision should be made through the anterior rectus abdominis fascia lateral to the fusion line of the external oblique aponeurosis with that fascia. If this is not done, suture of Cooper's ligament and the transversalis fascia will be completed under excessive tension.

The repair is completed by imbricating the external oblique aponeurosis either superior or inferior to the spermatic cord. A personal preference is to combine an over and under approximation, tie the lower flap beneath and the upper flap over the cord. Careful attention to normalize the new subcutaneous inguinal ring to facilitate the patient's passing future employment examinations should be exercised.

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A METHOD OF GASTROSTOMY USING A LOOP OF JEJUNUM*

JOSEPH M. MILLER, M.D.†

Ft. Howard, Md.

Gastrostomy as a palliative procedure for malignant lesions producing esophageal obstruction has fallen into general disrepute since the results obtained from operation have been poor. A great number of operative technics have been

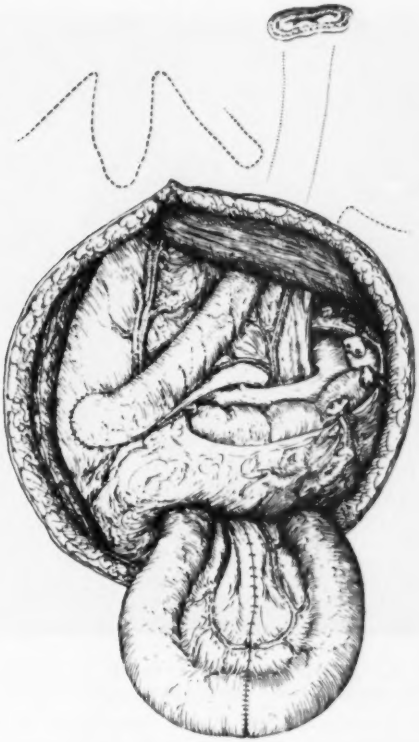

Fig. 1—Details of operation.

developed to avoid the complications inherent in each of these procedures. Although knowing the procedure is poor, when confronted with an inoperable

*Reviewed in the Veterans Administration and published with the approval of the Chief Medical Director. The statements and conclusions published by the author are the result of his own study and do not necessarily reflect the opinion or policy of the Veterans Administration.

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proximal obstructing lesion, the surgeon is tempted each time to perform a gastrostomy.

The risk of operation in such chronically ill, debilitated patients is considerable. Loss of weight, anemia and hypoproteinemia are usually severe and although attempts toward correction can be made, the underlying disease prevents restoration of the patient to normal. The addition of a gastrostomy in order to feed the patient has been the last answer for palliation or preparation of the patient for a definitive operation. Pneumonia, wound infection and an unsatisfac-

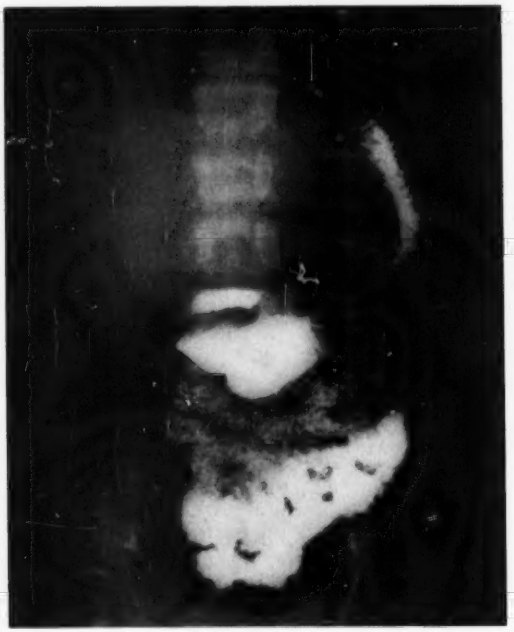

Fig. 2—Loop of jejunum is superimposed upon shadow cast by stomach. Note high implantation of cutaneous end of jejunum and the loop formed.

tory stoma producing leakage are the more frequent postoperative complications.

Despite discouraging results and a general pessimistic attitude, the problem of creating a means of feeding patients with obstruction proximal to the stomach must be accepted. Dissatisfaction with the usual methods of gastrostomy has prompted a trial of a method using an isolated loop of jejunum. A search of the literature showed that a similar operation had been done by Tavel[1] in 1906. The procedure to be reported is a modification of that reported by Tavel.

Case Report

W.J.C. (R-30587), a 35 year old white male, was admitted to the Veterans Administration Hospital, Fort Howard, Maryland on August 21, 1951 with an inoperable squamous-cell carcinoma of the larynx. There was progressive hoarseness and dysphagia. He also had diabetes mellitus of a severe degree which was difficult to control because of the inability to eat properly.

Through a paramedian incision with lateral retraction of the upper portion of the left rectus abdominis muscle, 22 cm. of jejunum with its blood supply intact were isolated starting just distal to the first major arterial arcade. An end-to-end jejunojejunostomy was done. Openings were made in the transverse mesocolon and in the gastrocolic omentum along the greater curvature of the stomach. The isolated portion of the jejunum was then passed upward to the anterior surface of the stomach. Three centimeters of the left seventh costal cartilage were

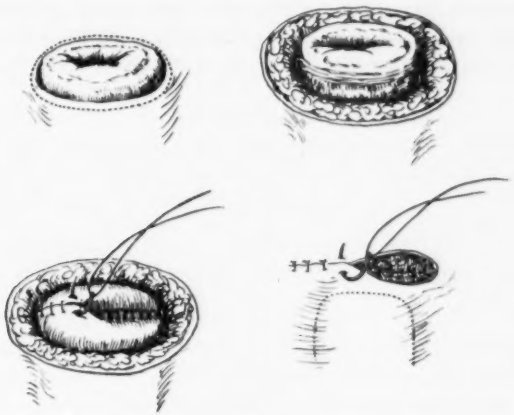

Fig. 3—Details of closure of cutaneous end of jejunum.

next removed and the peritoneum incised just below the costal cartilage. A subcutaneous tunnel was made from this area to a small transverse incision made midway between the left nipple and the costal margin. The loop of jejunum was threaded through the tunnel and arranged in an isoperistaltic manner to form a loop. An end-to-side jejunogastrostomy, using the anterior surface of the stomach then was done. Two Kocher forceps were used to hold the cutaneous end of the jejunum in place (Fig. 1).

The wound healed uneventfully although it was necessary to remove about two centimeters of redundant jejunum at the cutaneous margin ten days later. Feedings were started through the tube (Fig. 2) on the same day. The patient chewed his food and then expectorated into a funnel connected to a rubber tube placed within the loop of jejunum. The diabetes mellitus was now more easily

controlled. He received roentgen therapy for the carcinoma of the larynx and was discharged from the hospital on December 21.

Comment

Leakage and regurgitation were never a problem because of the isoperistaltic position of the tube and the trap formed by the loop. Pain in the loop of jejunum or in the surrounding area was never noted and other evidence of a jejunal ulcer was not seen.

Closure of the new opening will be easy if regression of the malignancy producing the esophageal obstruction ever permits. Under local anesthesia, the proximal portion of the jejunal loop can be isolated and inverted (Fig. 3). Placed in a subcutaneous position, it can be reopened and used if necessary.

The procedure described differs from that of Tavel in that the loop of jejunum was not brought out through the operative wound but through a separate incision with the loop of jejunum being carried up on the chest. The operation is not difficult to do and provides a leak-proof means of feeding.

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PRESIDENTIAL REPORT

WILLIAM W. LERMANN, M.D.

Pittsburgh, Pa.

At this Annual Meeting of the National Gastroenterological Association, I wish to express my thanks and appreciation to all of you for your loyalty to, and interest in the National Gastroenterological Association during the past year. This will be the last official meeting I shall conduct as your President and I assure you that the past year has been an interesting and busy one for all your officers and National Council members.

During the past twelve months many things have been accomplished and changes have been brought about.

Our biggest problem, sufficient and adequate help to assist the Executive Officer, has been partially solved by authorizing additional compensation and personnel for his office staff.

The second problem which occupied our attention for a good part of the year was the membership drive. A letter, brochure and application blank was sent to all members and the results are promising. The drive for additional members will be continued this coming year.

Other administrative problems which arose from time to time were solved and it is hoped that these changes will make for a better, more cohesive organization.

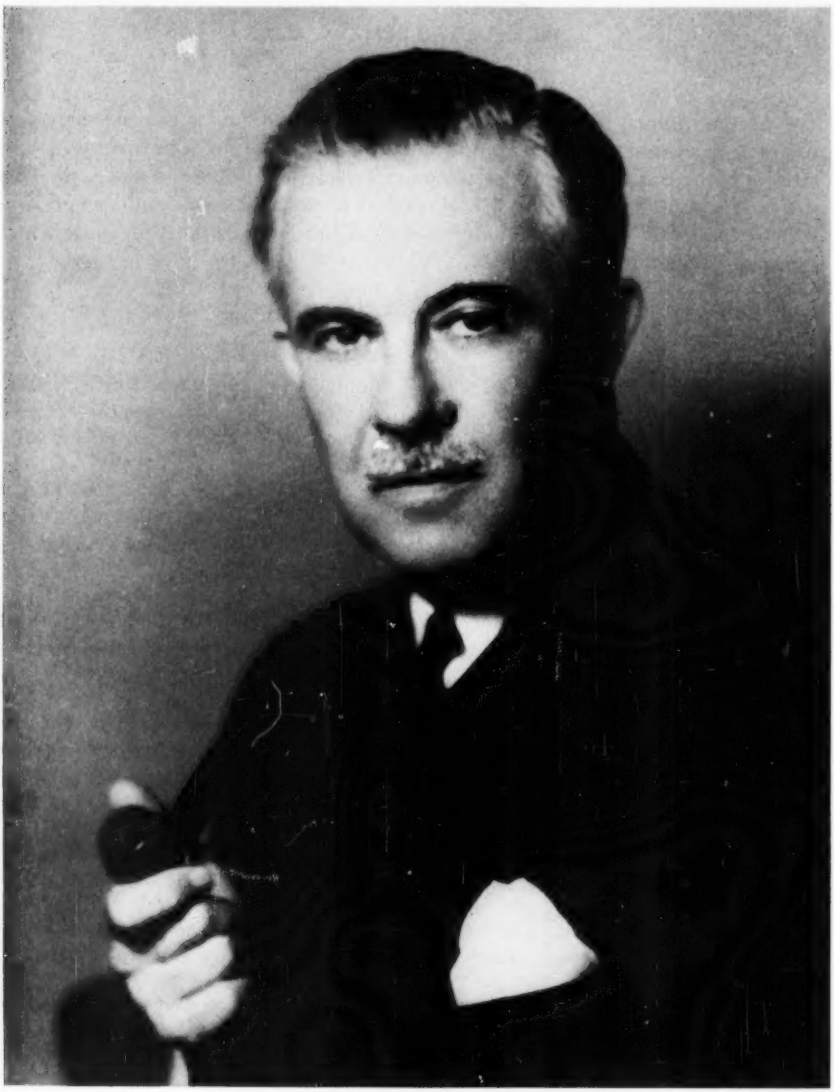
Changes in the Constitution and By-Laws have been considered by the Executive Committee and the National Council, which have as their purpose a closer integration of the members at large in the organization. More about these changes will be brought to your notice in the coming year.

We are striving to improve our official publication, THE REVIEW OF GASTROENTEROLOGY and to this end, a new editorial board was appointed.

For the first time this year, we established a permanent publicity committee to publicize our Convention, Postgraduate Course and other activities.

A Ladies' Auxiliary has been established and it is hoped that it will be functioning in time for our Los Angeles Convention in 1953.

Your National Council feels that your Association has made considerable progress; has a more business-like administration and that a definite effort has been made to increase advantages to all members, thus making membership in the Association more worthwhile. Once again, I hope that each one of you will immediately, upon returning home, make a concerted effort to obtain from



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WILLIAM WALSH LEHMANN, M.D.
Past President

amongst the outstanding colleagues in your community, at least two new members to help swell the ranks of this fine Association.

In passing on the responsibility of the Presidency to Dr. Felix Cunha of San Francisco, I feel that he will not only carry through the new projects already begun, but no doubt will have much to offer in new plans for the coming year. This will not be an easy task for him, as he must come from San Francisco every 3 months to attend meetings in New York. He must, in addition, devote a great deal of time to the position. I would venture to say that in the next 12 months, your incoming President will be obliged to devote to the duties of his office at least one-quarter of his working hours.

Once again, may I express my sincerest thanks for your assistance and loyalty during this past year and extend my personal good wishes to each and every one of you.

CHAPTER ACTIVITIES

Boston Chapter

A meeting of the Boston Chapter of the National Gastroenterological Association was held at the Boston City Hospital on Thursday, 6 November 1952.

A panel discussion on "Jaundice" was presented by Dr. Chester Jones, Dr. Francis Moore and Dr. I. R. Jankelson.

Officers of the Chapter for the year 1952-1953 are: President, Dr. Henry Baker; Secretary-Treasurer, Dr. Charles W. McClure; Recording Secretary, Dr. John J. Byrne.

New Jersey Chapter

The New Jersey Chapter of the National Gastroenterological Association met in Newark on 13 November 1952.

The speakers were Dr. Alexander Strelinger, Dr. S. M. Gilbert and Dr. W. F. Vogel.

For the year 1952-1953, the officers of the Chapter are: President, Dr. Arthur Statman; Vice-President, Dr. Benjamin Macchia; Secretary, Dr. Leonard Troast.

New York Chapter

At the Annual Meeting of the New York Chapter of the National Gastroenterological Association, officers elected for 1952-1953 were: Dr. Harry Barowsky, President; Dr. Frederic W. Bancroft and Dr. Franz J. Lust, Vice-Presidents; Dr. A. X. Rossien, Secretary; Dr. William Ostrow, Recording Secretary and Dr. Elihu Katz, Treasurer.

On Monday, 10 November 1952, the Chapter held its first meeting of the year at the New York Academy of Medicine. The speakers were Dr. Charles L. Fox, Jr. and Dr. Walter Modell.

NEWS NOTES

Executive Committee Meetings

Executive Committee meetings of the National Gastroenterological Association were held at the Hotel Statler in New York City on 15 June and 14 September 1952.

At the meeting held on 15 June 1952, following reports of various committees, the Secretary-General, Dr. Roy Upham reported that the Boston, New Jersey, New York and Rhode Island Chapters had held regular meetings.

The Treasurer, Dr. Elihu Katz, reported on the financial status of the Association.

Dr. Upham suggested, and the committee unanimously voted to have the Treasurer's report distributed to each member of the committee at each meeting.

The Los Angeles, Memphis and New York Chapters presented for ratification the applications for membership of the following, which applications were approved by them and accepted by the Executive Committee: Dr. John William Perry, Hollywood, Calif.; Dr. Marion L. Rice, Memphis, Tenn.; Dr. James H. Licht, New York, N. Y.; Dr. Julius Rosenberg, Brooklyn, N. Y. and Dr. Leon Sasson, New York, N. Y.

The following were elecetd to membership at large in the National Gastroenterological Association: Dr. Carlton F. Brown, Detroit, Mich.; Dr. Richard E. Dunkley, Washington, D. C.; Dr. Francis Theodore Lytle, Fargo, N. D.; Dr. Marcus H. Sugarman, Detroit, Mich.; Dr. Clayton K. Stroup, Flint, Mich.; Dr. Robert R. Sterling, Detroit, Mich.; Dr. Albert L. Steinbach, Detroit, Mich.; Dr. Homer Martin Smathers, Detroit, Mich.; Dr. Earl G. M. Krieg, Detroit, Mich.; Dr. Lyle E. Heavner, Grosse Pointe, Mich.; Dr. Paul J. Connolly, Detroit, Mich.; Dr. William S. Carpenter, Detroit, Mich.; Dr Lewis Carbone, Detroit, Mich.; Dr. Duncan A. Cameron, Detroit, Mich.; Dr. J. Kenner Bell, Detroit, Mich.; Dr. L. E. Beeuwkes, Detroit, Mich.

Upon presentation of the necessary qualifications, the following were advanced as indicated: Dr Abraham I. Brenner, Brooklyn, N. Y., Fellow; Dr. Tomas C. Durante, Montevideo, Uruguay, Associate Fellow; Dr. Walter Freymann, West New York, N. J., Fellow; Dr. Bertram W. Gifford, Saugerties, N. Y., Associate Fellow; Dr. Hans J. Joseph, Richmond Hill, N. Y., Associate Fellow; Dr. Jacob M. Leavitt, New York, N. Y., Associate Fellow; Dr. Michael A. Michaels, Manchester, N. H., Associate Fellow; Dr. Joseph Roger Van Dyne, Forest Hills, N. Y., Associate Fellow and Dr. Saul A. Schwartz, New York N. Y., Fellow.

Dr. Samuel Weiss, Editor of The Review of Gastroenterology, reported on the financial status of the publication. Dr. C. Wilmer Wirts reported for the

committee appointed to study improvements in the journal. The report was tabled for further discussion.

Dr. C. J. Tidmarsh, chairman of the committee on Constitutional Revision was not present and in his absence, Dr. Johnsen moved that the changes to be made in the Constitution be tabled at this time.

One change, however, was approved for submission to the Annual Meeting which consisted of an amendment to the By-Laws to insert therein that members must be in good standing in the AMA, their county or state societies, which was inadvertently omitted from the Constitution.

Dr. Lynn Ferguson reported on the activities of the publicity committee.

The selection of the Edgewater Beach Hotel in Chicago for the 1955 Convention was unanimously approved. Upon motion made by Dr. Upham and seconded by Dr. Bassler, it was unanimously voted to return to the Hotel Statler in New York City for the 1956 Convention. The Executive Officer was empowered to arrange the dates.

The committee was advised that the New York Chapter planned a Twentieth Anniversary Banquet and it was unanimously voted to invite the Chapter to join with the National Association in celebrating this event.

A committee consisting of Drs. Katz, Johnsen and Upham was appointed to make arrangements for the affair. Drs. Bassler and Jacobson were designated to coordinate the plans of the National Association and the New York Chapter.

At the meeting held on 14 September 1952, routine administrative matters were disposed of.

Dr. Katz, the Treasurer, presented a written statement which was given to each member of the committee.

The Louisiana, New Jersey, New York, Philadelphia, Pittsburgh and San Francisco Chapters presented for ratification the applications for membership of the following, which applications were approved by them and accepted by the Executive Committee: Dr. Sidney G. Mack, Baton Rouge, La.; Dr. Ferdinand G. Weisbrod, East Orange, N. J.; Dr. Alexander Zabin, Malverne, N. Y.; Dr. Stanley Stark, Brooklyn, N. Y.; Dr. Bertram F. Moore, New York, N. Y.; Dr. Arnold L. Berger, Elmont, N. Y.; Dr. Herman Miller, Philadelphia, Pa.; Dr. N. Keith Hammond, Pittsburgh, Pa.; Dr. William B. Hetzel, Pittsburgh, Pa.; Dr. Abraham Bernstein, San Francisco, Calif. and Dr. Mortimer Weiss, San Francisco, Calif.

The following were elected to membership at large in the National Gastroenterological Association: Dr. John R. Tambone, Woodstock, Ill.; Dr. Isidore I. Hirschman, Huntington, W. Va.; Dr. Allen P. Newman, Fremont, Ohio; Dr. William A. Leonard, Jr., Oil City, Pa.; Dr. Claude G. Hooten, Clearwater, Fla. and Dr. I. M. Riffin, Upper Montclair, N. J.

Upon presentation of the necessary qualifications, the following were advanced as indicated: Dr. Abe Alper, Far Rockaway, N. Y., Associate Fellow; Dr. William E. Bippus, West Palm Beach, Fla., Fellow; Dr. Ludmilla F. Brown, New York, N. Y., Fellow; Dr. Francesco D'Imperio, Camden, N. J., Fellow; Dr. Arden S. Turner, Philadelphia, Pa., Fellow.

Dr. Weiss presented a financial statement for THE REVIEW OF GASTROENTEROLOGY.

Dr. Bassler reported that the New York Chapter had agreed to hold a joint banquet with the National Association in celebration of the Twentieth Anniversary and that a concerted effort to obtain a large attendance would be made. He further reported that a symbol of the office of President, consisting of a medallion on a ribbon, was suggested by Dr. Franz Lust. This medallion would be used in the formal induction of the new President of the Association.

Upon motion made, seconded and carried, it was voted to have such a medallion made and further, to invite all past Presidents of the Association to be present at the Twentieth Anniversary Banquet and participate in the ceremony.

Dr. Ferguson reported on the publicity for the Convention and Postgraduate Course.

Dr. Johnsen proposed the appointment of a committee to study the advisability of forming a Ladies' Auxiliary. The President was instructed to take this matter under advisement and make whatever appointments were deemed necessary.

With the consent of the Executive Committee, Dr. William E. Bippus of West Palm Beach, Fla., was appointed a member of the nominating committee for a period of 3 years.

It was decided to suspend the prize award contest for the time being and to study the possibilities for restoring the contest at a future date.

A finance committee consisting of Drs. Frank Cummings, Sigurd W. Johnsen and Elihu Katz presented recommendations concerning the Annual Budget of the Association for the year.

They also recommended that the Treasurer sign all checks which must be countersigned by one other officer. The consolidation of all accounts was another recommendation.

Upon motion duly made, seconded and carried, it was voted to submit an amendment to the By-Laws at the Annual Meeting in October, providing for the Tresaurer to sign checks issued for THE REVIEW OF GASTROENTEROLOGY.

The question of consolidating all accounts was tabled as being impractical at the present time.

Annual Meeting of the Executive Committee

The Annual Meeting of the Executive Committee of the National Gastroenterological Association was held at the Hotel Statler in New York City on 19 October 1952.

The Association was advised of the death of Dr. Seweryn S. Cytronberg, formerly of Mexico City, a Fellow of the Association.

Dr. Roy Upham, Secretary-General, reported that there were 21 chapters in the Association with a total chapter membership of 515.

He further reported that the New York, New Jersey and Boston Chapters had held meetings and had elected new officers for the year 1952-1953.

Dr. Elihu Katz, Treasurer, submitted a financial statement for the Association.

Dr. Bruce Lockwood, chairman of the membership committee, reported a total membership of 788 and that 101 new members had been elected to the Association in the past year.

The Milwaukee, New York and Pittsburgh Chapters presented for ratification the applications of the following, which applications were approved by them and accepted by the Executive Committee: Dr. M. C. F. Lindert, Milwaukee, Wisc.; Dr. Albert G. Schutte, Milwaukee, Wisc.; Dr. Henry Colcher, New York, N. Y. and Dr. William W. McNaugher, Pittsburgh, Pa.

The following were elected to membership at large in the National Gastroenterological Association: Dr. Dan Zavela, East Detroit, Mich. and Dr. Joseph V. Petrelli, New Haven, Conn.

Dr. Samuel Weiss, Editor, presented a financial statement for The Review of Gastroenterology.

Dr. Arthur A. Kirchner, speaking for the editorial policy committee, made a preliminary report and recommendation.

Dr. Lermann, reporting for the program committee, advised that the program was an excellent scientific presentation and hoped that it would be well received.

Dr. Lynn A. Ferguson reported that the committee on publicity had prepared abstracts of the papers being presented, for the use of the lay press. He further reported on the extent of the publicity for the Convention and Postgraduate Course.

Dr. William C. Jacobson advised that the program for the banquet had been arranged and that several acts of entertainment would be on the program.

The chairman of the Constitutional Revision Committee, Dr. Tidmarsh, presented the draft of the amendments to the Constitution and By-Laws by his com-

mittee which would make for a more cohesive organization. These recommendations were approved and were to be submitted to the National Council at its meeting immediately following.

Dr. William W. Lermann, retiring President, appointed with the consent of the Executive Committee, an administrative advisory committee consisting of Drs. Sigurd W. Johnsen, C. Wilmer Wirts and A. Xerxes Rossien. This committee was to assist the Executive Officer in problems which might confront him in relation to the administration of the affairs of the Association.

A rising vote of thanks was given Dr. Lermann for his services as President of the Association.

Annual Meeting of the National Council

The Annual Meeting of the National Council of the National Gastroenterological Association was held at the Hotel Statler in New York City on Sunday, 19 October 1952.

The Council heard the reports of the various committee chairmen and ratified the actions of the Executive Committee meetings held during the interim between Council meetings.

The recommendations of the Executive Committee concerning the amendments to the Constitution and By-Laws submitted by the committee on Constitutional Revision were accepted. The Council approved the submission of these changes to the membership for action at the Annual Meeting to be held in Los Angeles in 1953.

By unanimous vote, Dr. Hyman I. Goldstein of Camden, N. J., historian of the Association was elected to Honorary Fellowship.

A Ladies' Committee, appointed by Dr. Lermann, consisting of Mrs. Felix Cunha, Mrs. Arthur A. Kirchner, Mrs. Sigurd W. Johnsen and Mrs. Samuel Weiss were meeting to consider the possibility of forming a Ladies' Auxiliary.

Annual Meeting of the National Gastroenterological Association

The Annual Meeting of the National Gastroenterological Association was held at the Hotel Statler in New York City on Monday, 20 October 1952.

Dr. William W. Lermann, presided and presented the President's report which appears elsewhere in the journal.

The various committee chairmen gave their reports and the Treasurer presented an audited financial statement for the calendar year ending 31 December 1951.

The Secretary reported that no additional nominations had been received and upon motion duly made, seconded and carried, he was instructed to cast one vote for the slate presented by the nominating committee.

Two amendments to the By-Laws, which were submitted for action, were uanimously adopted.

Dr. Lermann then presented Dr. Felix Cunha, the incoming President who briefly spoke to those assembled.

Seventeenth Annual Convention

The attendance at the Seventeenth Annual Convention of the National Gastroenterological Association surpassed that of the last few Conventions and this was in no small measure the result of the excellent scientific program presented.

The Symposium for the General Practitioner was well attended and received, as were all of the other sessions throughout the 3 days.

The scientific exhibits, as well as the commercial and technical exhibits attracted many persons who expressed their appreciation of the efforts of the exhibitors.

On Monday evening, the Fourth Annual Convocation Ceremony took place, at which over 100 certificates were presented to new and advanced members of the Association. Participating in the ceremony were the officers of the Association and those who received their certificates. The invocation was delivered by Rabbi Leo Jung of the Jewish Center in New York and the benediction by Rev. John O. Mellin of the First Presbyterian Church of New York. Dr. Andrew A. Eggston, President-Elect of the Medical Society of the State of New York welcomed the members and guests to the city.

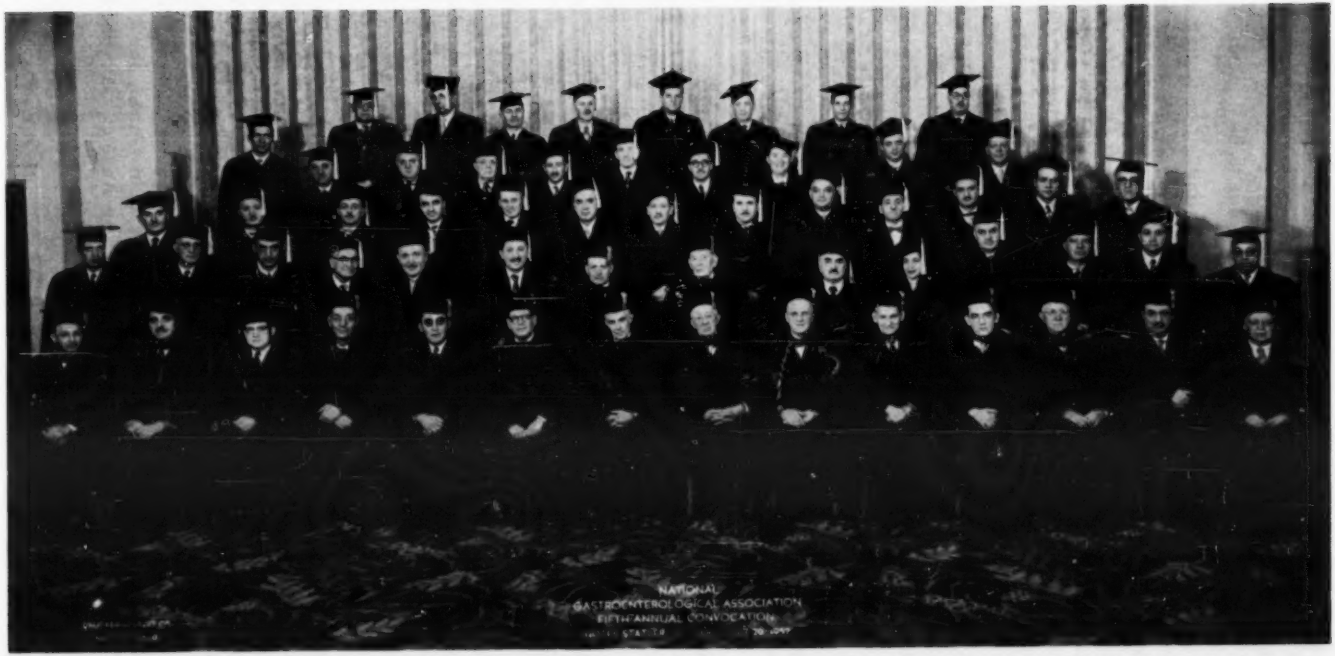
Honorary Fellowship certificates were presented to Dr. Abraham Ayala Gonzalez of Mexico City and Dr. I. Snapper of New York who delivered the principal address on "The History of Clinical Teaching of Medicine".

Following the convocation, members and guests attended the President's reception which again was sponsored by Winthrop-Stearns Inc.

Twentieth Anniversary Banquet

On Tuesday evening, 21 October 1952, the National Gastroenterological Association held its Annual Banquet at the Hotel Statler in New York City.

This marked the Twentieth Anniversary of the Association which was originally incorporated in New York State in November, 1932. The affair was a joint celebration with the New York Chapter, which was the original Society for the Advancement of Gastroenterology. The invocation was delivered by Rev. John Graham of the Church of St. Jean de Baptiste of New York.



Part of the group who participated in the Fifth Annual Convocation at the Hotel Statler in New York City on 20 October 1952. In the first row are, from left to right: Dr. A. X. Rossien; Dr. Arthur A. Kirchner; Dr. Lynn A. Ferguson; Dr. I. Snapper; Dr. Abraham Ayala Gonzalez; Dr. Felix Cunha; Dr. William W. Lermann; Dr. Anthony Bassler; Dr. C. J. Tidmarsh; Dr. Sigurd W. Johnsen; Dr. James T. Nix; Dr. Roy Upham; Dr. Elihu Katz; and Dr. Samuel Weiss. In the second row, seventh and eighth from the left are Dr. Andrew A. Eggston and Dr. G. Randolph Manning.

Present at the banquet were Drs. I. Ritter, G. Randolph Manning, Anthony Bassler and C. J. Tidmarsh, former Presidents of the Association who were given Past President's keys in recognition of their services. Drs. W. R. Morrison and H. W. Soper, two other former Past Presidents were unable to be present.

For the first time that evening, the President's medallion, consisting of a sterling silver replica of the Association seal, surrounded by a wreath and suspended from a red ribbon, was used in the induction of the new President, Dr. Felix Cunha of San Francisco. The medallion was given to Dr. Ritter and it was then placed in the hands of each succeeding President until Dr. Tidmarsh placed it around Dr. Lermann's neck. Dr. Lermann, in turn, invested Dr. Cunha with the insignia and Dr. Lermann received the Past President's key from Dr. Cunha.

Joey Adams, famed Broadway night club entertainer and comedian was master of ceremonies. He brought with him several acts for the entertainment of those present.

Course in Postgraduate Gastroenterology

The Course in Postgraduate Gastroenterology of the National Gastroenterological Association followed the Seventeenth Annual Convention in New York City.

The sessions on 23, 24, 25 October 1952 at the Hotel Statler and at The Mt. Sinai Hospital were again under the competent direction of Dr. Owen H. Wangensteen, who served as surgical co-ordinator and Dr. I. Snapper, who served as medical co-ordinator.

Over 100 physicians from all parts of the United States and several from foreign countries attended the Course. The faculty was chosen from amongst the eminent members of the staffs of the medical schools in New York City.

The next Course in Postgraduate Gastroenterology will be given immediately following the Eighteenth Annual Convention in Los Angeles, Calif., and will again be directed by Drs. Wangensteen and Snapper.

Ladies' Auxiliary

A Ladies' Auxiliary of the National Gastroenterological Association was organized at the Annual Convention held in New York City in October. A preliminary committee, consisting of Mrs. Felix Cunha, Mrs. Sigurd W. Johnsen, Mrs. Arthur A. Kirchner and Mrs. Samuel Weiss, called a luncheon meeting of the ladies attending the Convention, 14 of whom were present.

After determining that a Ladies' Auxiliary was necessary, the following officers were elected for the year 1952-1953: President, Mrs. Felix Cunha, San Francisco, Calif.; Vice-President, Mrs. Arthur A. Kirchner, Los Angeles, Calif.; Secre-

tary, Mrs. Sigurd W. Johnsen, Upper Montclair, N. J. and Treasurer, Mrs. F. H. Voss, Phoenicia, N. Y.

Mrs. Kirchner and Mrs. Cunha will be in charge of preparations for the ladies attending the 1953 Convention in October. There will be no dues charged and the doctors' wives will receive information concerning the activities prior to the Convention.

Those interested in the Ladies' Auxiliary are asked to communicate with Mrs. Johnsen, the Secretary.

Executive Committee Meeting

A meeting of the new Executive Committee of the National Gastroenterological Association was held at the Hotel Statler in New York City on Wednesday, 22 October 1952.

Mrs. Sigurd W. Johnsen submitted a written report of the initial luncheon meeting of the Ladies' Auxiliary, which had been held at the Statler on the previous day. The report gave the officers and stated the plans for next year.

Dr. A. X. Rossien, Secretary, requested permission to bring the biographical data files in the office up to date by sending out questionnaires to the membership. This request was approved.

The dates for the next Executive Committee meetings and the Semi-Annual meeting of the National Council were tentatively set.

It was unanimously voted to send extracts of the minutes of the National Council meeting, which pertain to the proposed amendments of the Constitution and By-Laws, to the entire Fellowship of the Association for their consideration.

Dr. Cunha, with the consent of the Executive Committee, appointed the standing committees and several special committees, continuing for the time being the two special committees appointed by his predecessor, Dr. Lermann. These were the Editorial Policy Committee and the Administrative Advisory Committee.

Appointment of Committees

Dr. Felix Cunha, President of the National Gastroenterological Association, with the consent of the Executive Committee, has anounced the appointment of the following standing committees:

Program Committee:—Dr. Anthony Bassler, New York, N. Y., Honorary Chairman; Dr. Felix Cunha, San Francisco, Calif., Chairman; Dr. Sigurd W. Johnsen, Passaic, N. J.; Dr. Arthur A. Kirchner, Los Angeles, Calif.; Dr. Lester M. Morrison, Los Angeles, Calif.; Dr. Ralph Scovel, San Francisco, Calif.; Dr. Roy Upham, New York, N. Y. and Dr. Samuel Weiss, New York, N. Y.

Membership Committee:—Dr. Frank A. Cummings, Providence, R. I., Chairman; Dr. John E. Cox, Memphis, Tenn.; Dr. Boston Day, San Francisco, Calif.; Dr. Bruce C. Lockwood, Detroit, Mich.; Dr. C. J. Tidmarsh, Montreal, Canada and Dr. Fred H. Voss, Phoenicia, N. Y.

Editorial and Publication Committee:—Dr. Samuel Weiss, New York, N. Y., Chairman and Editor of THE REVIEW OF GASTROENTEROLOGY; Dr. C. Wilmer Wirts, Philadelphia, Pa. and Dr. Arthur A. Kirchner, Los Angeles, Calif.

Endowment Fund Committee:—Dr. William C. Jacobson, New York, N. Y., Chairman: Dr. Elihu Katz, New York, N. Y.; Dr. William W. Lermann, Pittsburgh, Pa. and Dr. C. Wilmer Wirts, Philadelphia, Pa.

Research Committee: Dr. C. Wilmer Wirts, Philadelphia, Pa., Chairman, the balance of the committee to be chosen later.

Convention Committee:—Dr. Samuel Weiss, N. Y., Chairman and Dr. Elihu Katz, New York, N. Y.

Committee on Postgraduate Education:—Dr. William W. Lermann, Pittsburgh, Pa., Chairman; Dr. Ernest Fishbaugh, Los Angeles, Calif.; Dr. I. Snapper, Chicago, Ill. and Dr. Roy Upham, New York, N. Y.

Nominating Committee:—The nominating committee is provided for by the By-Laws and this year consists of Dr. Felix Cunha, San Francisco, Calif.; Dr. Sigurd W. Johnsen, Passaic, N. J.; President and President-Elect respectively; Dr. G. Randolph Manning, New York, N. Y.; Dr. E. A. Marshall, Cleveland, Ohio; and Dr. William E. Bippus, West Palm Beach, Fla.

The following special committees were also appointed:

Committee on Constitutional Revision:—Dr. C. J. Tidmarsh, Montreal, Canada, Chairman; Dr. William C. Jacobson, New York, N. Y.; Dr. Sigurd W. Johnsen, Passaic, N. J.; Dr. Arthur A. Kirchner, Los Angeles, Calif.; Dr. A. X. Rossien, Kew Gardens, N. Y. and Dr. Roy Upham, New York, N. Y.

Public Relations Committee:—Dr. Lynn A. Ferguson, Grand Rapids, Mich., Chairman; Dr. E. Forrest Boyd, Los Angeles, Calif.; Dr. Arthur A. Kirchner, Los Angeles, Calif.; Dr. Leigh Watson, Los Angeles, Calif. and Dr. Mortimer Weiss, San Francisco, Calif.

Committee on Finance and Budget:—Dr. William W. Lermann, Pittsburgh, Pa., Chairman; Dr. Sigurd W. Johnsen, Passaic, N. J. and Dr. Elihu Katz, New York, N. Y.

For the time being, the following special committees appointed by Dr. Lermann were continued.

Editorial Policy Committee:—Dr. Arthur A. Kirchner, Los Angeles, Calif., Chairman; Dr. Bruce C. Lockwood, Detroit, Mich. and Dr. C. Wilmer Wirts, Philadelphia, Pa.

Administrative Advisory Committee:—Dr. Sigurd W. Johnsen, Passaic, N. J., Chairman; Dr. A. Xerxes Rossien, Kew Gardens, N. Y. and Dr. C. Wilmer Wirts, Philadelphia, Pa.

Election of Officers

At the Annual Meeting of the National Gastroenterological Association in New York City in October, the following officers of the Association were elected for the year 1952-1953: President, Dr. Felix Cunha, San Francisco, Calif.; President-Elect, Dr. Sigurd W. Johnsen, Passaic, N. J.; First Vice-President, Dr. Lynn A. Ferguson, Grand Rapids, Mich.; Second Vice-President, Dr. James T. Nix, New Orleans, La.; Third Vice-President, Dr. Arthur A. Kirchner, Los Angeles, Calif.; Fourth Vice-President, Dr. C. Wilmer Wirts, Philadelphia, Pa.; Secretary-General, Dr. Roy Upham, New York, N. Y.; Secretary, Dr. A. Xerxes Rossien, Kew Gardens, N. Y. and Treasurer, Dr. Elihu Katz, New York, N. Y.

Members of the National Council elected for 4 years are: Dr. Frank J. Borrelli, New York, N. Y.; Dr. H. Necheles, Chicago, Ill.; Dr. Louis L. Perkel, Jersey City, N. J.; Dr. C. J. Tidmarsh, Montreal, Canada and Dr. Fred H. Voss, Phoenicia, N. Y.

Eighth International Cruise-Congress of the Pan-American Medical Association

The Pan-American Medical Association will hold its Eighth International Medical Cruise-Congress aboard the S. S. "Nieuw Amsterdam", sailing from New York on 7 January 1953.

Dr. Henry A. Rafsky of New York, Fellow of the National Gastroenterological Association, has been elected a Vice-President of the Pan-American Association.

Prize for Paper on Diabetes By Medical Students and Interns

The American Diabetes Association offers a $250.00 prize to *medical students* and *interns* for a paper on any subject relating to diabetes. The paper can be a report of original studies, a biographical or historical note, a case report with suitable comment, or a review of the literature.

Manuscripts must be submitted on or before April 1, 1953 to the Editorial offices of *Diabetes:* The Journal of the American Diabetes Association, 11 West 42nd Street, New York 36, New York. The papers will be reviewed by the Editorial Board, which will take into consideration the value of the material and method of presentation in selecting the best paper.

The award of $250.00 has been made possible through the generosity of the St. Louis Diabetes Association, an Affiliate of the American Diabetes Association.

Postgraduate Course In Diabetes and Basic Metabolic Problems

The first Postgraduate Course in Diabetes and Basic Metabolic Problems to be conducted by the American Diabetes Association will be offered under the direction of Charles H. Best, C.B.E., M.D., F.R.S., Director of the Banting and Best Department of Medical Research of the University of Toronto, on January 19, 20, 21, 1953, at the University of Toronto, Canada.

Developed by the Association's Committee on Postgraduate Education, under the chairmanship of Edward L. Bortz, M.D., the Course will have as its Clinical Director, Ray F. Farquharson, M.B., Professor of Medicine of the University of Toronto, and Andrew L. Chute, M.D., Professor of Pediatrics of the University of Toronto, will act as Associate Clinical Director.

Over thirty lectures and round-table discussions have been planned as well as a social evening. The Course is open to non-member physicians as well as members of the American Diabetes Association, but the number of registrants will be limited to 100. Fees are $20 to members, $40 to non-members. Details of the three-day program and registration and hotel information may be obtained from J. Richard Connelly, Executive Director, American Diabetes Association, 11 West 42nd Street, New York 36, N. Y.

Errata

In the article "Physiologic Basis for the Therapeutic Effects of Cortisone" by Dr. Samuel Soskin which appeared on pages 808 to 816 of the October, 1952 issue, several illustrations were inadvertently transposed.

Figure 2 on page 810 should have been Figure 6; Figure 3 on page 811 should have been Figure 5; Figure 5 on page 814 should have been Figure 3 and Figure 6 on page 815 should have been Figure 2.

The corrections have been made in the author's reprints.

In Memoriam

We record with profound sorrow the passing of Dr. Seweryn S. Cytronberg, Fellow, of New York City and Dr. W. G. Colvin, Member, of Grand Rapids, Mich.

Our deepest sympathies are extended to the bereaved families.

ABSTRACTS

GASTROINTESTINAL TRACT

LYMPHOSARCOMA OF THE GASTROINTESTINAL TRACT: Ira M. Lockwood. J.A.M.A., 150:435-437, (Oct. 4), 1952.

Surgery is indicated in malignant growths of the gastrointestinal tract, however, in the radiosensitive group, lymphoblastoma, x-ray therapy as an adjunct is indispensable.

Lymphosarcoma, the commonest lymphoid-tissue tumor of the gastrointestinal tract occurs as polypoid projections into the lumen of the bowel, or as metastatic growths in the mesenteric nodes; the former as primary tumors and the latter from more generalized neoplastic disease.

Two types of cells can be recognized, the reticulum cell (large round cell), and the malignant lymphocytoma (small round cell), their presence in the submucosa, involving the nerve plexuses accounts for the early colicky pain.

Other signs of malignancy: loss of weight, mild secondary anemia, sometimes palpable abdominal masses and gradual (never acute) intestinal obstruction occur as in any other abdominal malignancy. Achlorhydria is common, but melena is rare, occurring only after the tumor has reached a large size and necrosis has developed.

In the stomach differentiation from carcinoma is difficult, except that the tumor occurs most frequently in the proximal portion of the organ and among the younger age groups.

In the small bowel, also difficult of diagnosis, lymphosarcoma is situated in the terminal ileum, early pain being the important symptom. However, in contrast to carcinoma, the involved area shows irregular dilatation rather than constriction, the mucosal pattern being destroyed, and the lesion being more elongated than is found in carcinoma.

In the colon the tumor simulates carcinoma, by presenting a polypoid appearance on x-ray, and thus is usually diagnosed until operation.

Rectal lymphosarcoma can be recognized early by biopsy.

Under radiation therapy many of the lymphosarcomas undergo rapid regression, even though of large size before treatment.

Surgical extirpation of the primary tumor with involved nodes plus postoperative irradiation seems to be a most satisfactory method of present day therapy.

Diagnostic laparatomy is justified when lyphosarcoma is suspected, even though operative removal be questioned roentgenologically, because these tumors tend to remain in the intestinal mucosa and metastasize relatively late as compared with carcinoma.

Metastatic spread is through the lymphatics, hence radiation is indicated over entire area of involvement as well as anticipated areas of involvement.

Under the combined present day therapy, cecal lymphosarcoma has the highest five-year cure rate, despite frequent recurrence in the small bowel lesions.

The rectal type of this disease invariably recurs, even under adequate treatment, however, the new growth are highly radiosensitive and can easily be shrunken by x-ray, thus relieving obstruction and improving the patient clinically.

Nitrogen mustards and radioisotopes have to date been unsuccessful in treating lymphosarcoma of the gastrointestinal tract.

J. E. Brown

CONTRIBUTION TO THE STUDY OF THE ETIOLOGY OF THE HIATAL HERNIA AND THE DIVERTICULOSIS OF THE DIGESTIVE TRACT: M. Brembart, G. Coupatez and Y. Laurent. Arch. mal. de l'app. dig. 41:412 (Apr.), 1952.

A case of Ehlers-Danlos' disease associated with a hiatal hernia, gastric diverticulum, duodenal diverticulum, diverticulosis coli and sideropenic anemia.

In a previous clinical and roentgenological study on hiatal hernia, the authors pointed out the fact that in many cases the etiology of this disease remains obscure.

A recent case of particularly complex pathological association observed by the authors seems to furnish some new elements about the possible etiology, not only of the hiatal hernia, but also the diverticulosis of the digestive tract and the sideropenic anemia, both so often associated with the hiatal hernia.

The diagnosis of "generalized elastic fibrodysplasia", called Ehlers-Danlos' syn-

drome, can be made with certainty.

This is a congenital disease of dominant character considered as a systemic disease affecting the mesenchymatous tissue.

The authors believe that in this case the hiatal hernia as well as the diverticula of the digestive tract are in direct relation with the general cause of the Ehlers-Danlos syndrome, that means hyperelasticity of the mesenchymatous system extended to the fibromuscular structure of diaphragmatic hiatus as well as the submucus layers of the digestive tube.

On the other hand, hemorrhagic diathesis without difficulties of coagulation is present in about 25 per cent of cases of the Ehlers-Danlos disease.

This congenital fragility of the blood vessels perhaps explains the high frequency of anemia associated with hiatal hernia.

ESOPHAGUS

ESOPHAGEAL VARICES: Marcel Brombart. Acta Gastroenterol. Belg. 14:637. (July-Aug.), 1951.

Esophageal varices constitute an element of great importance in the picture of many diseases, which are associated with portal hypertension. The early demonstration by a roentgenological examination is of considerable diagnostic importance. The roentgenological examination, in order to be most successful has to rest not only on anatomical and anatomicropathological findings, but also should take in account physiological and pathophysiological factors, which influence the visibility of the varices. In spite of the confusion arising from the many other possible affections, or roentgenological aspects, the diagnosis is generally easy, as the site of most varices is in the lower thoracic segment of the esophagus. The illustrations of this article are excellent and most instructive.

FRANZ J. LUST

SPONTANEOUS RUPTURE OF THE ESOPHAGUS: S. A. Mackler. Surg. Gynec. & Obst. 95:345-356, (Sept.), 1952.

Spontaneous rupture of the esophagus is an accident which may follow a sudden rise in intraabdominal pressure, usually vomiting. Other acts which have resulted in rupture are: Straining at defecation, lifting a heavy weight, a convulsive seizure of grand mal epilepsy, and the labor of child birth. Distinction is made between rupture and perforation of the esophagus. The triad of vomiting, low thoracic pain, and emphysema of the neck, is diagnostic and constitutes sufficient evidence to warrant a left thoracotomy. Evidence is presented to support the conclusion that rupture occurs in a previously normal healthy esophagus as a result of intraesophageal pressure transmitted from the stomach. The rupture uniformly occurs at the lower extremity of the esophagus upon the left lateral wall, apparently the weakest part of the organ. That this is indeed the weakest part of the esophagus was confirmed by experimentally inflating and bursting the esophagi of 65 fresh human cadavers. The site and configuration of the resulting rupture duplicated exactly to clinical lesion. Three cases of spontaneous rupture of the esophagus were presented. The treatment was surgical: Left thoracotomy, suture of the esophagus, and drainage. The recovery of two of the patients brings to a total of thirteen, those surviving a spontaneous rupture of the esophagus. The outcome was uniformly fatal before the adoption of a surgical method of treatment.

J. R. VAN DYNE

BENIGN ULCER OF THE ESOPAGUS ASSOCIATED WITH DUODENAL ULCER. ITS RELATION WITH ESOPHAGEAL DYSKINESIAS: M. Brombart, Y. Laurent, J. Godart. Arch. mal. de l'app. dig. No. 5, 1952.

The authors report on a case of peptic ulcer of the esophagus associated with duodenal ulcer and esophagus dyskinesia in a 39 year old man.

The roentgen diagnosis was confirmed by clinical findings, endoscopy, operation and later evolution.

The noted dyskinesia was made manifest by secondary contractions which originated in the median ⅓ of the esophagus, spreading either downwards or in cephalic direction (antiperistaltism).

The peptic ulcer of the juxta-diaphragmatic segment of the esophagus showed typi-

cal roentgenological appearance. The duodenal ulcer was of the scarred type.

The general condition of the patient was improved by gastrectomy, which had to be completed by esophagus dilatations producing the disappearance of the ulcer niche and stenosis.

Dynamic disorders of the esophagus were still observed one and half a years after the treatment was suspended.

The authors stress the extreme rarity of the esophagus peptic ulcer not associated with brachyesophagus and roentgenologically diagnosed. This rarity becomes particularly obvious when compared with the extreme frequency of duodenal ulcer.

The elements of the roentgen diagnosis of ulcer of the esophagus are exposed, its etiology and pathogeny discussed. Cardiospasms, which had been observed in their patient before appearance of the duodenal ulcer and the peptic ulcer of the esophagus, are supposed to have favored the formation of the latter.

The authors think that the esophagus dyskinesia, manifested by secondary contractions persisting one and half a years after the clinical and radiological healing of the narrowing of the esophagus and the ulcer, are in relation to irreversible changes of the intraparietal nervous components, owing to the chronic inflammatory process in the neighborhood of the ulcer.

THE GASTROESOPHAGEAL REFLUX: M. Brombart and R. van Lerberghe. Acta Gastroenterol. Belg. 15:66, (Feb.), 1952.

The authors assume that many diseases and functional troubles of the esophagus are still not well known, especially the gastroesophageal reflux. They notice that the etiology and the pathology of this physiopathological phenomenon, as well as its relation to pyrosis, are still obscure. They agree with others upon the necessity of separating the gastroesophageal reflux from vomiting, regurgitation and rumination, and of determining criteria of the reflux. The study of reflux provocated by "siphonage" (swallowing of water in supine position) permitted obtaining some unpublished roentgenological appearances. The reflux was found in 51 cases, 21 of which had a hiatus hernia. Cases with short esophagus did not show the reflux or only very rarely. Paraesophageal herniae are often associated with the reflux. The authors are very careful in making definite conclusions, but are continuing their studies.

FRANZ J. LUST

STOMACH

LOCAL RECURRENCE FOLLOWING SUBTOTAL RESECTION FOR GASTRIC CARCINOMA: S. B. Thomson and R. A. Robins. Surg. Gynec. & Obst. 95:341-344, (Sept.), 1952.

In those patients with carcinoma of the stomach who develop recurrence in the immediate lymphatic drainage area after partial gastrectomy, it is more likely that these glands were already the site of microscopic secondaries at the time of operation, and that they became involved because of secondary spread from subsequent recurrence in the gastric or duodenal stumps. The gastric stump recurrence rate following partial gastrectomy for carcinoma of the stomach is approximately 30 per cent. The duodenal stump recurrence rate following partial gastrectomy for carcinoma of the stomach is 10-15 per cent. This would be significantly reduced if the whole of the first portion of the duodenum were routinely resected in all operations for carcinoma of the stomach. A significant portion of the immediate lymphatic drainage of the stomach lies in the peripancreatic tissues and is technically inseparable from the pancreas. This commonly involved portion of the immediate lymphatic drainage is not removed in radical partial gastrectomy as at present carried out for gastric carcinoma. For this reason there is local recurrence in this area in over 50 per cent of cases. This peripancreatic portion of the immediate lymphatic drainage of the stomach could only be excised by adding total pancreaticoduodenectomy to the radical operative procedure in carcinoma of the stomach. In pyloric carcinoma that remains reasonably localized in the growth, subtotal gastrectomy plus radical excision of the immediate lymphatic drainage is more likely to improve the cure rate than is routine total gastrectomy.

J. R. VAN DYNE

RESULTS OF RECORDED DUODENAL INTUBATION CHECKED BY RADIOMANOMETRY: G. Albot, G. F. Bonnet, J. Toulet and H. Dressler. Arch. mal de l'app. dig. 41:No. 2, (Feb.), 1952.

For nearly three years, using recorded duodenal intubation with a study of the flow, the double Meltzer-Lyon test (using pure oil with magnesium sulfate) and the novocaine test suggested by Varela Lopez, Varela Fuentes and Martinez Prado, the authors have concentrated on checking whether the standard interpretations were borne out by the radiomanometric and anatomicopathological findings.

Grouping their observations under three headings, according to whether the intubation revealed isolated disturbances in vesicular time or in Oddian time or even simultaneous disturbances of both vesicular and Oddian time, the authors conclude:

1. Diagnosis of vesicular stasis is easily attributed to the notion of hyperconcentration of the B bile, verified by chemical dosing. The etiological diagnosis of this stasis is more difficult, and the standard diagrams are not always in agreement with one another.

The syndrome of intubation characterized by an erratic, inadequate and interrupted vesicular flow with distinct pains during the interruptions is practically typical of *cystic hypertension*.

But at the other extreme, a copious, prolonged and painless vesicular flow with a copious and painless second response, considered by Varela Lopez as typical of *vesicular atonia*, may equally well be due to cystic hypertension. Furthermore, all observations of intubation with *isolated disturbances of vesicular time*, and of whatever type these disorders may be, are shown to be connected with *cystic hypertension*, and most frequently to have a mechanical (valve, partitioning) lipoidic (strawberry gallbladder) or adenomatous origin (Rokitanski-Ashoff sinus).

2. Isolated disturbances of the time of the Oddi are exceptional (except in the case of cholecystectomized patients), and the prolongation of the time of the closed Oddi observed in four cases has never corresponded with a spasm or an odditis on verification by biliary radiomanometry.

In one case, the prolongation of the closed Oddi with definite action of the novocaine and normal vesicular time, was the only tell-tale sign of isolated cysticitis. This poses the problem of the physiological connection between the cystic and the Oddi.

In two cases the same syndrome was attributable to a disorder of biliary excretion (cirrhosis or icterus). It is likely that it is often a matter of a simple defect in the opening of the Oddi, and not of a spasm of the sphincter.

3. The vast majority of observations include *disturbances of both the time of the Oddi and that of the bladder*. But only exceptionally are disturbances of a similar type at the level of the Oddi found by radiomanometry (one case in eleven).

Among these varied results of intubation, some throw into relief the definite action of novocaine on the opening of the Oddi, and thus allow a deductive diagnosis of *cystic hypertension* to be made.

Elsewhere the novocaine is apparently *inactive* (Oddi's sphincter only opens quite a long time after its administration), and this phenomenon coincides with the existence of *vesicular atonia* or of an inflamed or sclerous *idiopathic acystolia* (without cystic obstruction).

4. The authors end by suggesting a physiological hypothesis which enables the phenomena noted and their divergences from the findings of the Uruguayan authors to be explained.

A CLINICAL AND PATHOLOGICAL STUDY OF THE SIGNIFICANCE OF MALIGNANT PYLORIC OBSTRUCTION ON POSTRESECTION PROGNOSIS: A. D. Anderson, M. B. Dockerty and H. K. Gray. Surg. Gynec. & Obst., 95:85-92, (July), 1952.

Included in the study were the cases of 100 patients having clinically obstructing carcinoma of the pyloric end of the stomach with retention of more than 500 c.c. of gastric contents who underwent resection with the hope of a cure between 1940 and 1944 inclusive. Partial gastrectomy was performed in 99 cases and total gastrectomy in 1 case with a hospital mortality rate of 8 per cent. Their analysis suggests the following conclusions:

1. Contrary to common belief, gross pyloric obstruction by itself is indicative of a poorer postresection prognosis than is indicated in the absence of obstruction.

2. A history of short duration of symptoms suggests the course of the disease is rapidly progressive while long duration of

symptoms with or without ulceration is more hopeful.

3. From a pathological standpoint a grossly infiltrating lesion, Borrmann's type III or IV, is of serious significance; a localized lesion, type I or II is of favorable significance.

4. Prognosis is favorably influenced by the microscopic (Broder's) grade of the lesion.

J. R. VAN DYNE

OBSERVATIONS ON THE VIGOROUS DIAGNOSTIC APPROACH TO SEVERE UPPER GASTROINTESTINAL HEMORRHAGE: E. D. Palmer. Ann. Int. Med., 36:1484, (June), 1952.

In a military situation, the author studied one hundred twenty-one patients for acute, severe, upper gastrointestinal hemorrhage, without previous history of bleeding. The routine followed was treatment of shock together with immediate diagnostic procedures of esophagoscopy, gastroscopy and radiography. In no instance could it be said that the manipulation aggravated the bleeding.

The diagnosis was established during the first few days in seventy-eight per cent. In order of frequency the most common causes of bleeding were duodenal ulcer, hypertrophic gastritis, gastric ulcer, and erosive gastritis.

The advantages of early diagnosis as a guide to treatment are obvious and outweigh the theoretical disadvantages of aggravating the bleeding. This experience strongly suggests a more vigorous diagnostic approach to the problem of massive gastrointestinal hemorrhage of undetermined origin.

H. B. SILBERNER

GASTRIC CARCINOMA, A STATISTICAL STUDY, 1938-1947: E. E. Jemerin and R. Colp. Surg. Gynec. & Obst. 95:99-112, (July), 1952.

It was found that the duration of symptoms prior to diagnosis and treatment was overlong. In 54 per cent of the cases it was more than 6 months, with no diminution in the second 5 year period as compared with the first. Ulcer was a common cause of diagnostic delay. Resectability increased from 41 to 52 per cent largely through technical advances and extension of operative limits. Mortality for subtotal gastrectomy decreased from 28 to 12.6 per cent despite the extension of surgery. The employment of total gastrectomy was extended and resection of cardiac and lower esophageal lesions added. The mortality of these while reduced is still appreciable. The 3 year survival rate in terms of percentage of resection survivors increased from 33.3 per cent in the first 5 year group, to 42.9 per cent in the second 5 year group. The 5 year rates of survival for the same periods were 23.8 per cent and 27.3 per cent.

A further increase in survival might be obtained by employing radical subtotal or total resection routinely even for antral and fundal lesions. However, even within the first 6 months of symptoms a large proportion of cases already showed irremediable lesions signifying very rapid invasion or a long silent period. Major improvement in survival depends upon a higher percentage of resectable cases. This can only result from marked diminution in the delay before treatment with diagnosis in the silent interval as the ultimate goal.

J. R. VAN DYNE

FREQUENCY AND EVOLUTION OF CANCERIZED GASTRIC ULCER: M. Demole and P. Michaud. Arch. mal. de l'app. dig. 41:393, (April), 1952.

The lack of precise criteria on cancerous transformation of peptic ulcer is one of the reasons why this subject is so often discussed.

By using repeated barium test meals, using Gutmann's "radioclinical" method, one comes nearer to an exact diagnosis; but even the surgeon, at gross examination, cannot be sure of the kind of ulcer he feels under his fingers, so that the histological criterion—although discussed—is the only certain way, proving the presence of a previous true peptic ulcer that has recently undergone a cancerous transformation.

But using this method, one can only report on gastric resections and necropsies.

On 495 stomachs removed for gastric ulcer, the authors found 69 cancerous transformations, i.e. 13.9 per cent. This very high proportion agrees with 7 other statistics published since 1945: 405 cancerized ulcers on 2,845 operated gastric ulcers, or 14.25

per cent. This does not mean that every seventh gastric ulcer is going to result in a cancer, as only chronic ulcers are removed. This great number, however, should encourage the physician to send their patients who do not heal under medical treatment more often and sooner to the surgeon.

Anamnestic details were obtained on 51 of these 69 patients. The majority (28) had a typical history of peptic ulcer, lasting up to 20 years. In most of them, the symptoms had changed recently, enough to attract notice; but sometimes, the clinical picture changed slowly, as in the other ordinary complications of peptic ulcers.

The minority (23) of these cases complained either of dyspepsia, or of epigastric pain with no ulcerous characteristics. These pains did not last as long as those of the first group (6 months to 2 years), and their cancerous nature was more often suspected.

The prognosis for these cancerized ulcers is not good, even when the malignant part was very small, and discovered only by histological examination.

INTESTINES

LYMPHOSARCOMA OF THE SMALL INTESTINE: J. W. Faulkner and M. B. Dockerty. Surg. Gynec. & Obst. 95:76-84, (July), 1952.

Thirty-three cases of surgically treated lymphosarcoma of the small intestine were studied. The patients almost without exception had symptoms referable to the abdomen and characteristically suggestive of chronic intestinal obstruction. Hematemesis, melena, or diarrhea was not seen frequently and a history characteristic of sprue was elicited in only 2 cases in which there was marked intestinal involvement. Loss of weight was present in all but 2 cases and could be expected to be significant in any case in which the symptoms were present for more than two months. The presence of a mass was noted in 19 cases. Laboratory studies were of no specific value and in none of the cases was a leukemic picture found. Roentgenologic examination presented positive evidence of a lesion in 21 cases. The pathologic examination revealed that the growth assumes one of three major gross forms: Polypoid, aneurysmal, or ulcerative. Treatment consisted of efforts at wide removal and adequate node bearing mesenteric resection. Postoperative roentgen therapy was used in practically all cases. Of 14 patients surviving operation and eligible for 5 year study, only 3 survived for 5 or more years.

J. R. Van Dyne

SURGICAL TREATMENT FOR IDIOPATHIC CONGENITAL MEGACOLON (Hirschsprung's Disease): D. State. Surg. Gynec. & Obst. 95:201-212 (Aug.), 1952.

Physiological study of the colon, as determined by fluoroscopic and x-ray examination after the administration of a barium enema, in 15 patients with congenital megacolon revealed: a. A normal appearing rectum; b. A narrow segment of the rectosigmoid portions of the colon of variable extent; c. Marked dilatation and absence of peristaltic waves and normal haustral markings of the descending, sigmoid, and variable portions of the left side of the transverse colon; d. Active peristalsis and normal haustral markings of the cecum, ascending, and right side of the transverse colon. When the colon was exposed at laparotomy the differences between the right and left side of the colon are marked and correspond to the roentgenographic findings. A one stage transabdominal operative procedure is described by the author in which, the narrowed terminal segment of the sigmoid and portions of the colon showing no peristaltic activity roentgenographically are removed. The residual part of the large bowel (ascending and right side of the transverse colon) is anastomosed to the upper portions of the rectum at a distance of six to ten centimeters from the anal skin margin.

The operation described has been done in 16 patients without a death. Fifteen had excellent results while the remaining patient has had to resort to enemas on several occasions since operation. A period of follow-up in these cases ranges from six to thirty-six months.

The saving of the rectum is of importance, because it eliminates the question of damage to the nervi erigentes which is a deterrent to the whole-hearted acceptance of these operative procedures in which the rectum is removed.

This operative procedure has also been applied to two patients with megarectum and pseudomegacolon. Although both pa-

tients developed a leak at the line of anastomosis, necessitating proximal colostomy, subsequently, when the colostomies were closed, they both developed daily spontaneous bowel movements.

J. R. Van Dyne

CARCINOMA OF THE RECTUM IN THE FEMALE: C. D. Knight, J. N. Waugh, M. B. Dockerty. Surg. Gynec. & Obst. 95:220-228 (Aug.), 1952.

A comparative study of two surgical series of carcinomas of the rectum in female patients are presented. These series consisted of carcinomas involving the anterior wall of the rectum, but in one series all the lesions, in addition, were accompanied by involvement of the posterior vaginal wall which necessitated partial vaginectomy at the time of abdominoperineal resection. The result of the study allowed the following conclusions to be drawn: 1. There is no significant difference in the prognosis for a female patient with an operable rectal lesion confined to or involving the anterior quadrant, and the prognosis as reported in other surgical series of rectal carcinomas located at the same level.

2. Physical examination of patients who have rectal carcinoma gives inconclusive information concerning the local extent of malignant process. Clinically, inflammatory fixation may be indistinguishable from cancerous fixation, even in the presence of rectovaginal fistulae.

3. The incidence of actual malignant invasion from the vagina grossly adherent to a rectal carcinoma was 64.9 per cent.

4. Patients whose vaginal involvement is purely on the basis of inflammation have a slightly better prognosis than those in whom malignant vaginal extension has occurred.

5. Complete circumferential growth of the carinoma is not necessary before extrarectal and vaginal extension occur.

6. Growth involvement of the vagina by a rectal malignant process is a valuable prognostic sign. Patients with such involvements have less than half the survival of patients with similar lesions but without fixation.

7. The existence of a malignant rectovaginal fistula is of ominous prognostic importance. No patient in this series who had this complication lived five years.

8. Involvement of the lymph nodes is a more reliable index of prognosis than is local extension without lymphatic spread.

9. An overall five year survival rate of 26.8 per cent and an operative mortality rate of about 5 per cent justify and make wide removal of the posterior vaginal wall imperative when it is adherent to a carcinomous rectum.

J. R. Van Dyne

A NEW APPROACH FOR EXCISION OF CARCINOMA OF THE LOWER PORTION OF THE RECTUM AND ANAL CANAL: I. Sauer and H. E. Bacon. Surg. Gynec. & Obst. 95:229-240 (Aug.), 1952.

The factors which underly the difference in prognosis between low and high lying cancer of the rectum are discussed. Study of the anatomy of the pelvic fascia and of the distribution of the lymphatics of the rectum, show the classic description by Miles to be inaccurate and that it led to erroneous conclusions. As a result of the general acceptance of this description the lateral area of spread was not evaluated accurately nor properly excised. The necessity for re-evaluation of the anatomy of the pelvis was stressed, and a procedure for the excision of the area of such spread is presented in detail.

Results obtained cannot serve as a basis for final judgment. Still, they follow the sequence of ideas which lead the author to the point of view elaborated in the article.

Lesions located within the abdominal cavity can be cured by procedures that remove the superior area of spread which is exclusively intraabdominal. Extraperitoneal lesions, however, are drained as well by the extraperitoneal lymphatics and for that reason the same type of procedure applied here failed to accomplish its end. The classic abdominoperineal procedure developed by Miles is not sufficiently radical for the treatment of this type of lesion.

Instead of freeing the rectum from the fascia by cutting through the lateral ligaments, the proper procedure is to separate the fascia intact from the pelvic walls. The portion of the fascia which covers the pelvis is an important lymph-bearing area and should be removed. In addition to the wider removal of the fascia, the iliac vessels and their branches should be dissected in order to more completely eradicate the lymph bearing tissues.

J. R. Van Dyne

CONGENITAL HERNIA OF THE DIAPHRAGM WITH SPECIAL REFERENCE TO RIGHT-SIDED HERNIA OF THE LIVER AND INTESTINES: E. E. Arnheim. Surg. Gynec. & Obst. 95:293-307 (Sept.), 1952.

The surgical pathology and treatment of the most common types of congenital diaphragmatic hernia in infancy and childhood are reviewed. An analysis of reported cases of congenital right-sided diaphragmatic hernia of the liver and intestines treated surgically is presented. A technic for the plastic repairs of almost complete absence of a hemi-diaphragm utilizing the hernial sac is described. Three cases of congenital right-sided diaphragmatic hernia of the liver and intestines successfully treated surgically are reported.

J. R. Van Dyne

A COMPARISON OF CECOSTOMY AND TRANSVERSE COLOSTOMY IN COMPLETE COLON OBSTRUCTION: J. H. Alvers, and L. L. Smith. Surg. Gynec. & Obst. 95:410-415 (Oct.), 1952.

The conclusions arrived at were that surgery is often too long delayed in complete destruction of the colon. Expectant treatment with suction and enema is hazardous in large bowel obstruction, since in the majority of cases obstruction is of a closed loop type due to a competent ileocecal valve. Delay results in a more critically ill patient and the likelihood of cecal perforation; such perforation was noted in ten of the 146 patients undergoing operation in the present series. A flat plate of the abdomen and an emergency barium enema are roentgenologic procedures that aid in proper diagnosis.

Exploration, even though limited in certain cases, will obviate a missed diagnosis. Six deaths in this series were due to unknown causes of obstruction. Included were two patients with volvulus of the sigmoid who were treated by cecostomy. One patient with a perforated cecum was unsuccessfully treated by a transverse colostomy. Decompression of a severely distended bowel at the time of surgery not only improves that patient's chances of surviving but also makes the surgeon's work easier.

In the study, the tube cecostomy proved to be an inadequate emergency decompressing procedure. An exteriorized cecostomy was better. When there is a choice of procedures, a transverse cecostomy results in lower mortality, more effective decompression, less tendency to chemical inbalance, and earlier and better preparation of the colon for subsequent surgery. However, an exteriorized cecostomy is indicated in cases of perforation and partial gangrene of the cecum. It is also recommended for complete obstruction involving the ascending colon, hepatic flexure, and transverse colon. A transverse colostomy is recommended in diverticulitis of the left colon with complete obstruction and perforation, and incomplete obstructions of the rectum, sigmoid, and descending colon (other than volvulus).

Local anesthesia in poor risk patients, judicious use of blood transfusion, correction of chemical inbalance, and the use of antibiotics have made possible a more aggressive surgical approach in the presence of acute colon obstruction.

J. R. Van Dyne

ACUTE ADHESIVE ILEUS: W. F. Eecker. Surg. Gynec. & Obst. 95:472-476 (Oct.), 1952.

The concept that acute intestinal obstruction due to adhesions can be safely and effectively treated by nonoperative tube decompression, presupposes the existence of absolute diagnostic criteria for differentiating simple and strangulated obstructions. An analysis of 412 cases of acute adhesive ileus indicates that the absence or presence of fever, tachycardia, leucocytosis, a palpable mass, and peritoneal irritation, does not suffice to differentiate these two varieties of obstruction. Acute intestinal obstruction due to old adhesions should be treated by early operation. The preoperative preparation of the patient by suction applied to a long intestinal decompression tube, and the correction of fluid, electrolyte, and blood volume deficits can usually be effected in a period of less than six hours. Intestinal obstruction due to recent adhesions, will usually respond to suction, but conservative therapy must be employed with great caution and, unless evidence of recovery is prompt and unequivocal, operation is in order. The mortality rate in this series of 412 of acute adhesive ileus was 11.8 per cent. In the 52 cases in which tube decompression was abused, the mortality was 38.4 per cent.

J. R. Van Dyne

WHIPPLE'S DISEASE: F. R. Russo. Arch. Int. Med. 89:600 (April), 1952.

The author presents two new cases and reviews the literature of the rare condition first described by Whipple in 1907 as intestinal lipodystrophy.

Because of difficulties in diagnosis, only eight of the total of 48 cases reported to date have been diagnosed before laparotomy or autopsy. Anatomically, the principal lesions are in the mesenteric lymph nodes, which become involved with lymphogranulomatosis, and in the tunica propria of the small intestine, which becomes laden with foamy macrophages and may contain dilated lymph channels. The clinical picture is one of progressive weakness, weight loss, diarrhea, and abdominal discomfort, usually in middle-aged men; with physical findings of emaciation, hypotension, and edema; and laboratory reports of anemia, hypoproteinemia, hypocalcemia, and steatorrhea. The differential diagnosis involves primarily those conditions producing a sprue-like syndrome. The pathogenesis is unknown, and treatment has been symptomatic, without altering the fatal prognosis.

H. B. Silberner

PATHOLOGY AND LABORATORY RESEARCH

MULTIPLE BALLOON KYMOGRAPH RECORDING OF THE EFFECT OF BANTHINE, BELLADONNA AND PLACEBOS ON THE UPPER INTESTINAL MOTILITY: William P. Chapman, Arthur B. French, Phyllis Hoffman, and Chester M. Jones. New England J. Med. 246:435 (March), 1952.

The comparative action of the oral administration of Banthine, 100 mg. tincture of belladonna of 0.4 and 0.6 c.c. and placebos on the motility of the upper small intestine has been studied in 31 healthy adult subjects. The changes in propulsion, total contractions (propulsive and nonpropulsive) and tone were determined by a multiple balloon-kymograph recording method. Banthine was found to cause a striking decrease in propulsion and total contractions and a slight and moderate decrease in tonus. Banthine acted more rapidly and inhibited motility to a greater degree than did tincture of belladonna in either dosage. Both drugs produced significantly greater effects than were observed after placebo administration. Dryness of the mouth was more noticeable with Banthine than with tincture of belladonna. Banthine administration was followed by an increase in heart rate, which was not observed in the tincture of belladonna tests. Although Banthine compares very favorably with tincture of belladonna in its inhibitory action on intestinal motility, it remains for long-term clinical studies to determine which agent is of greater therapeutic value in the sustained treatment of gastrointestinal disorders.

Franz J. Lust

INVESTIGATION OF HYPOGLYCEMIA IN GASTRECTOMIZED PATIENTS; STUDY OF GLYCEMIC VARIATIONS AFTER INGESTION OF FAT. Henri and Andre Monges, Ch. Guigou and Ph. Bouillin. Arch. mal. de l'app. dig. 5:1952.

The authors have made a study of the effect of the ingestion of fats on glycemia in gastrectomized patients (subtotal gastrectomy for ulcer). They give the results obtained with crude olive oil after a dose of 40 to 50 c.c. At the same time they have investigated the effects on the glycemia of nongastrectomized patients of the ingestion, on the one hand, and of the instillation, on the other hand, of the same quantity of oil into the duodenum. The glycemic doses were administered before the doses of oil and later every quarter of an hour during the first hour and every half-hour during the following two hours.

The following facts emerge from this research:

1. With ⅝ of the gastrectomized patients (11 times out of 17) the ingestion of oil brings about definite hypoglycemia (30 to 45 per cent) in which the peak occurs ½ to 1 hour after the commencement of the test.

2. With normal patients, the ingestion of oil does not noticeably modify the rate of blood sugar; on the contrary, direct administration into the duodenum of the fat by tubing, brings about almost consistently (11 times out of 13) a lowering of glycemia fairly similar to that obtained with gastrectomized patients after ingestion.

The recorded lowering of glycemia in gastrectomized patients after ingestion of oil, and with intraduodenal instillation of this substance in normal subjects is attributed by these authors to the massive liberation of the duodenojejunal hypoglycemic

hormone (La Barre's incretine). The slow gastric evacuation of the oil in normal subjects after oral administration explains the absence of notable variations in glycemia.

The authors show the importance of their investigation on the diet to be prescribed for gastrectomized patients.

DUCCI'S RED COLLOIDAL TEST: Guy Albot and M. Corteville. Arch. mal. de l'app. dig. 41:284, 1952.

This test, studied in 171 cases of hepatobiliary disorders along with Guy Albot's various instantaneous liver tests seemed to produce very different results from the latter.

While the so-called passage tests (water, galactose, hippuric acid), the tests of MacLagan, Gross, Hanger and Kunkel seemed specially sensitive to the existence of an acute diffuse parenchymatous hepatitis, the red colloidal test on the other hand, when it is clearly positive, strong and permanent (4 and 5) has always seemed to indicate the existence in the liver parenchyma of a conjunctive reaction of the same type as mesenchymatous hepatitis or cirrhosis.

It is interesting to include it in an "instantaneous liver test" especially because its results do not always agree with those of other flocculation tests.

Everything seems to proceed as if the persistent anomaly of the red colloidal test revealed, and that prematurely, the basic lesion, the structural reformation of the liver, the mesenchymatous reaction which is generally conclusive. The other variable and unstable modifications of the passage tests and other flocculation tests would reveal, however, the episodes in the evolutive wave of acute diffuse parenchymatous hepatitis.

From the practical viewpoint, there is no doubt that the red colloidal test, equally as well as bromsulfalein, enables, in the presence of icterus, enlarged monosymptomatic liver or of vague dyspeptic disorders, the tracking down of chronic mesenchymatous hepatitis and of cirrhosis whose stable irregularities very accurately emphasize the progressive and steady evolution. In the presence of cholecystic icterus, its strong and permanent positiveness arouses fear of the development of cholecystic cirrhosis.

LIVER AND BILIARY TRACT

THE MANAGEMENT OF ACUTE CHOLECYSTITIS: R. L. Mustard and H. R. Custer. Surg. Gynec. & Obst. 95:59-62 (July), 1952.

The management of 211 cases seen during a 10 year period is discussed. The general trend of treatment here has been toward aggressive conservatism with surgery reserved only for those patients showing signs of uncontrolled and spreading abdominal disease. With this type of approach early operation was necessary in only 36 patients (28 having cholecystectomies and 8 cholecystostomies) the other 172 patients were sent home clinically improved and with instructions to return at a later date for interval surgery. There was no mortality in those below 50, and 2.3 per cent in the group over the age of 50. From the reports of several large representative groups on early surgery for this disease entity, there has been a mortality of approximately 1.5 per cent for patients under 50 and between 5 and 6 per cent for those over 50 years of age. The dangers of surgery in the presence of acute inflammation with all its concomitant findings around the delicate anatomy of the biliary ducts, especially in the hands of partially trained surgeons are discussed.

J. R. Van Dyne

OBSTRUCTION OF THE COMMON BILE DUCT, VISCOSITY STUDIES: M. W. Eisenstein, H. Necheles, G. Asrow and L. Walker. Surg. Gynec. & Obst. 95:93-98. (July), 1952.

The rate of flow of solutions of varying viscosities through the common bile duct was determined in anesthetized dogs. The rate of flow diminished as viscosity increased, and, with the most viscous solutions, flow stopped entirely. In the dog, variations of tone of the duodenum and of the sphincter of Oddi often affected the rate of flow considerably. Viscous bile or viscid solutions may produce an obstruction in the common duct. Clinical cases are quoted which seem to prove this contention.

J. R. Van Dyne

BOOK REVIEWS

THE MUSCULOSKELETAL SYSTEM: A Symposium presented at the Twenty-Third Graduate Fortnight of the New York Academy of Medicine. Edited by Mahlon Ashford, M. D. 368 pages, 103 illustrations. The MacMillan Co., New York, N. Y., 1952. Price $6.50.

In addition to the foreword, opening address by Dr. Watson and the preface, the book is divided into 16 chapters. Seventeen well known clinicians contributed their clinical experience to make this volume an excellent reference for the general practitioner as well as the specialist.

DISEASES IN OLD AGE: Robert T. Monroe, M. D., Harvard University Monographs in Medicine and Public Health, No. 11. 407 pages. Harvard University Press, Cambridge, Mass., 1951. Price $5.00.

A clinical and pathological study of 7,941 individuals over 61 years of age is the basis of this monograph. Here the social worker and the physician will find valuable statistics relating to individuals who were patients on the medical service of the Peter Bent Brigham Hospital over a period of 30 years.

Studies were made of diseases of the nervous, cardiovascular, respiratory and gastrointestinal systems; diseases of the genitourinary organs, the blood, venereal and nutritional diseases, bones and joints and malignancies.

A summary of the medical findings and the needs of old people, if they are to get well, are worthwhile reading.

PRESCRIPTION FOR MEDICAL WRITING — A USEFUL GUIDE TO PRINCIPLES AND PRACTICE OF EFFECTIVE SCIENTIFIC WRITING AND ILLUSTRATION: Edwin P. Jordan, M.D., and Willard C. Shephard. 112 pages with 26 figures. W. B. Saunders Co., Philadelphia, Pa., 1952. Price $2.50.

A well written and documented little book dealing with the preparation of medical papers for lectures or publication in a scientific journal.

The reviewer, who has written many articles for publication and lectures, finds the advice given by the authors sound and enlightening.

It is recommended that physicians buy and read the book and follow the sound advice, even though at present they do not contemplate writing an article for publication.

THE BATTLE FOR MENTAL HEALTH: James Clark Moloney, M.D. 105 pages. Philosophical Library, New York, N. Y., 1952. Price $3.50.

A very interesting little monograph which should be carefully read by expectant mothers, nurses, obstetricians, pediatricians and supervisors of hospitals where maternity wards still maintain separate infantoriums.

Dr. Moloney's ideas and ideals may someday be adopted and both mother and child will be better for it.

PENICILLIN DECADE: Lawrence Wild Smith, M.D. and Ann Dolan Walker, R.N. 122 pages. Arundel Press, Washington, D. C., 1951. Price $2.50.

Since the discovery of penicillin by Fleming, physicians have utilized it to a great extent with more or less success. This mold is not a panacea for all infectious conditions. Growth is inhibited of the staphyloccus, gonococcus, meningococcus and corynebacterium diphtheriae and it might also be a useful antiseptic for application to infected wounds.

Observers however, reported untoward ef-

fects in some individuals after penicillin injections. The physician should be on the alert for these manifestations and be in a position to counteract them as soon as they appear. Among the reactions may occur fever, urticaria, chills, headache, facial flushing, generalized muscular pain and tenderness at the site of injection.

The reviewer recommends that this little book be carefully read by physicians as a guide to the use and abuse of penicillin therapy.

LIVER DISEASE — A CIBA FOUNDATION SYMPOSIUM: Edited by G. E. W. Wolstenholme, O.B.E., M.A., M.B., B.Ch., Consulting Editor, Sheila Sherlock, M.D., F.R.C.P. 249 pages with 112 illustrations. The Blakiston Co., Philadelphia, Pa., 1951. Price $5.00.

The Ciba Chemical Company who founded the Ciba Foundation, through its secretary, Dr. Wolstenholme and Dr. Sherlock, planned this international assembly to discuss a portion of the vast field of hepatic investigation. As the reader may notice, emphasis was given to recent trends in the etiology of chronic liver disease.

The participating members of this symposium come from various American and European clinics and have contributed an enlightening array of pertinent information which should be of value to all physicians.

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metabolism

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When the nutritional status is threatened, **TAKA-COMBEX** provides a dual action which assures adequate vitamin intake as well as proper absorption and utilization of carbohydrates. **TAKA-COMBEX** supplies important factors of the B complex plus Taka-Diastase,® one of the most potent starch digestants known. In addition the Kapseals also contain vitamin C.

In pregnancy, during illness and convalescence, and in the management of geriatric patients, this enzyme-vitamin combination is a most valuable dietary adjunct: the vitamins assist carbohydrate metabolism, while the enzyme not only facilitates starch digestion but also enhances absorption of vitamin B.

KAPSEALS® TAKA-COMBEX

Each Kapseal contains:

Taka-Diastase (Aspergillus Oryzae Enzymes)	2½ gr.
Vitamin B_1 (Thiamine Hydrochloride)	10 mg.
Vitamin B_2 (Riboflavin)	10 mg.
Vitamin B_6 (Pyridoxine Hydrochloride)	0.5 mg.
Vitamin B_{12} (Cyanocobalamin)	1 mcg.
Pantothenic Acid (As the Sodium Salt)	3 mg.
Nicotinamide (Niacinamide)	10 mg.
Vitamin C (Ascorbic Acid)	30 mg.
Liver Concentrate N.F.	0.17 Gm.
Liver Fraction No. 2 N.F.	0.17 Gm.

In bottles of 100 and 1000.

ELIXIR TAKA-COMBEX

Each teaspoonful (4 cc.) contains:

Taka-Diastase (Aspergillus Oryzae Enzymes)	2½ gr.
Vitamin B_1 (Thiamine Hydrochloride)	2 mg.
Vitamin B_2 (Riboflavin)	1 mg.
Vitamin B_6 (Pyridoxine Hydrochloride)	0.5 mg.
Pantothenic Acid (As the Sodium Salt)	2 mg.
Nicotinamide (Niacinamide)	5 mg.

In 16-ounce bottles.

MEDICAMENTA VERA

Parke, Davis & Company

DETROIT, MICHIGAN

1st Lieutenant Lloyd L. Burke U.S. Army Medal of Honor

THE RED KOREAN strongpoint had stalled our attack; Lieutenant Burke saw that a breakthrough must be made. Rallying 35 men, he crept close to the enemy bunkers. He laid down a grenade barrage. Then he ran forward to an exposed knoll and opened a one-man pitched battle. He turned a light machine gun into the Red position. He caught live enemy grenades in mid-air and threw them back. Once he killed three men with his pistol. Before sunset Lieutenant Burke and 35 men had defeated 300. The lieutenant says:

"Every day, men who fought in Korea are coming home. They're finding jobs—partly because they and you and I own nearly 50 billion dollars' worth of U.S. Defense Bonds. For Bond savings—which protect our families financially—are also building a great backlog of *national* prosperity. Reason enough for investing in Bonds—don't you agree?"

★ ★ ★

Now E Bonds earn more! 1) All Series E Bonds bought after May 1, 1952 average 3% interest, compounded semiannually! Interest now starts after 6 months and is higher in the early years. 2) *All maturing E Bonds* automatically go on earning *after maturity*—and at the new higher interest! Today, start investing in better-paying Series E Bonds through the Payroll Savings Plan where you work! Or inquire at any Federal Reserve Bank or Branch about the Treasury's brand-new Bonds, Series H, J, and K.

Peace is for the strong! For peace and prosperity save with U.S. Defense Bonds!

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A vagal blocking agent for peptic ulcer with LOW incidence of SIDE EFFECTS

PRANTAL* methylsulfate (diphenmethanil methylsulfate) is an effective anticholinergic agent for treatment of peptic ulcer. Pain, pyrosis, nausea, and other symptoms of this syndrome are rapidly relieved. Troublesome side effects seldom occur.

*T. M. Tablets 100 mg. q. 6 h.

Schering CORPORATION
BLOOMFIELD, N. J.
In Canada: Schering Corporation, Ltd., Montreal, Que.

PRANTAL
methylsulfate

PRANTAL

SCHERING

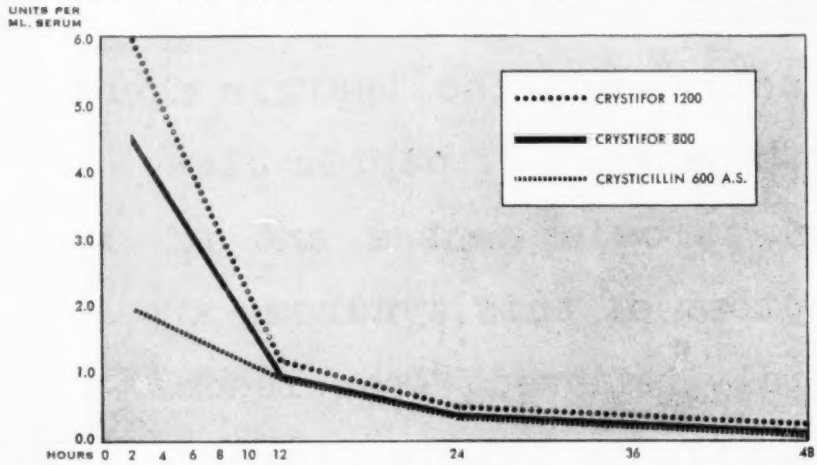
new *high potency* penicillin preparations

CRYSTICILLIN 600 A.S. Squibb Procaine Penicillin G, 600,000 units per 1.2 cc. *in aqueous suspension* ready to inject. Stable for 1 year if stored below 15 C. *10 dose silicone-coated vials (12 cc., 6,000,000 units), 1 dose 'Unimatic' disposable syringe.*

CRYSTIFOR 800 Squibb Procaine Penicillin G, 600,000 units, plus Potassium Penicillin G, 200,000 units, for aqueous injection. Injection volume 1.1 cc. *1 and 5 dose silicone-coated vials (800,000 and 4,000,000 units).*

CRYSTIFOR 1200 Squibb Procaine Penicillin G, 900,000 units, plus Potassium Penicillin G, 300,000 units, for aqueous injection. Injection volume 1.75 cc. *1 dose silicone-coated vials (1,200,000 units).*

SCHEMATIC SERUM CONCENTRATIONS OF PENICILLIN



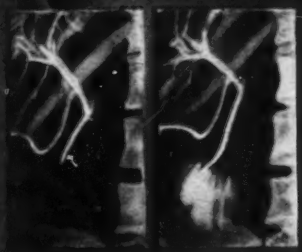
New antibiotic combinations also available:

DICRYSTICIN FORTIS, Squibb Procaine Penicillin G, 300,000 units, plus Potassium Penicillin G, 100,000 units, plus 1 Gm. Dihydrostreptomycin Sulfate, for Aqueous Injection. 1 dose vial. (Dicrysticin Fortis differs from Dicrysticin in that it contains twice the amount of dihydrostreptomycin — 1 Gm. instead of 0.5 Gm.).

DISTRYCILLIN A.S. Squibb Procaine Penicillin G, 400,000 units, plus 0.5 Gm. Dihydrostreptomycin Sulfate, in Aqueous Suspension. Stable for 1 year if stored below 15 C. Supplied in 1 and 5 dose vials (2 and 10 cc.).

SQUIBB A LEADER IN THE RESEARCH AND MANUFACTURE OF PENICILLIN AND STREPTOMYCIN

two sides to this story...



When spasm of the sphincter of Oddi (left) is relaxed (right), bile pours into the duodenum.

In many biliary conditions, combined hydrocholeretic and antispasmodic therapy is indicated for best results to flush the bile ducts with a greater volume of bile and to relax spasm in the sphincter of Oddi.

Cholan *hmb*

stimulation of free-flowing bile

relaxation of spasm in sphincter of Oddi

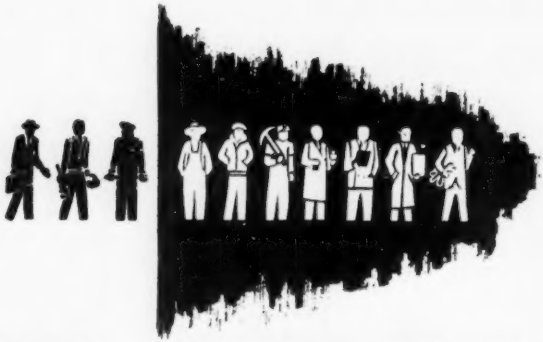
Dehydrocholic acid, the most potent hydrocholeretic known, stimulates copious secretion of thin, free-flowing bile... increases volume output by as much as 150%... is the least toxic of any bile salt, bile acid, or their derivatives.

*Homa*tropine *methyl*bromide and phenobarbital, by their synergistic spasmolytic-sedative actions, relax spasm of the sphincter of Oddi—and neutralize hypertonic dysfunction of the biliary tract.

Cholan-HMB contains, in addition to dehydrocholic acid-Maltbie, 250 mg. (3¾ gr.) per tablet, the spasmolytic *homa*tropine *methyl*bromide 2.5 mg. (1/24 gr.), and phenobarbital 8 mg. (⅛ gr.).

MALTBIE LABORATORIES, INC., Newark 1, N. J.

MALTBIE ... *first to develop American process for converting crude viscous ox bile into chemically pure dehydrocholic acid.*

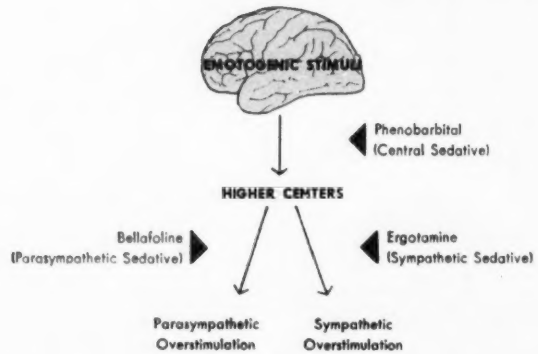


Anxiety . . . the universal disease

Relieve more anxious patients
with triple-acting

BELLERGAL®

Each tablet contains:
Bellafoline 0.1 mg. (1/650 gr.)
Ergotamine tartrate 0.3 mg. (1/210 gr.)
Phenobarbital 20 mg. (1/3 gr.)



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division of Sandoz Chemical Works, Inc.
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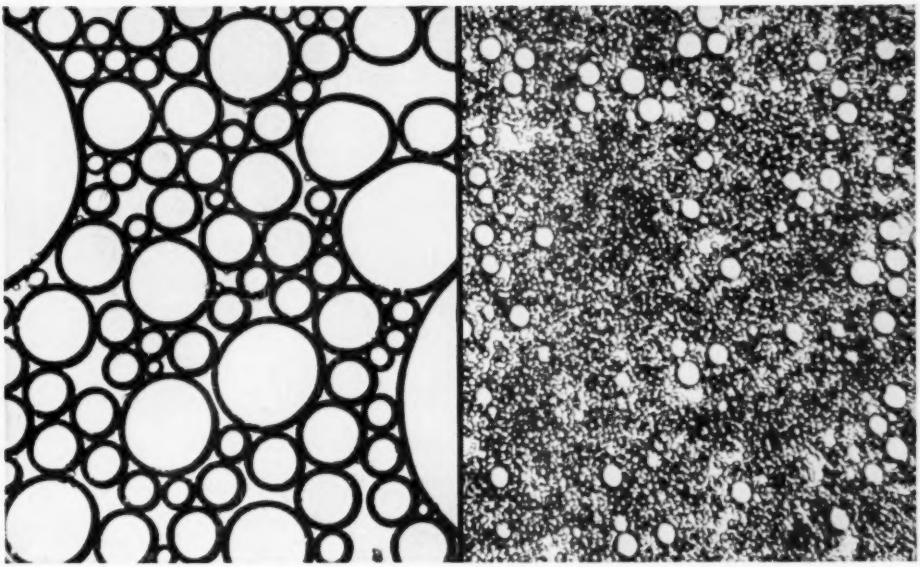


1 *Oil dispersion (x133). Large irregular globules fail to mix readily with fecal mass. Phenolphthalein is not evenly distributed to stimulate peristalsis. Action may be sporadic and evacuation incomplete.*

2 *The fine oil emulsion (x133) of Agoral. The small, uniform globules and the phenolphthalein mix readily with the bowel content, producing peristalsis by more uniform lubrication and stimulation.*

Which Laxative is Better—

COARSE DISPERSION OR FINE EMULSION?

Coarse dispersions are unstable, and erratic in their effects. Any physician can recognize the superiority of the fine Agoral emulsion (*at right, above*) compared with an ordinary oil-in-water dispersion (*left*).

Free-floating oil is distasteful and often regurgitated. Large oil globules tend to coalesce and form pools in the gut, which may seep past the sphincter as anal leakage.

Agreeable to Sensitive Stomach

The fine emulsion of Agoral is palatable and will not distress a sensitive stomach. It assures more uniform dosage and distribution of the active ingredients, more uniform clinical results.

Its thorough admixture with the bowel content gives effective, uniform lubrication of the fecal mass as well as the canal. There is no loose oil to cause anal leakage.

Mixed like Homogenized Milk

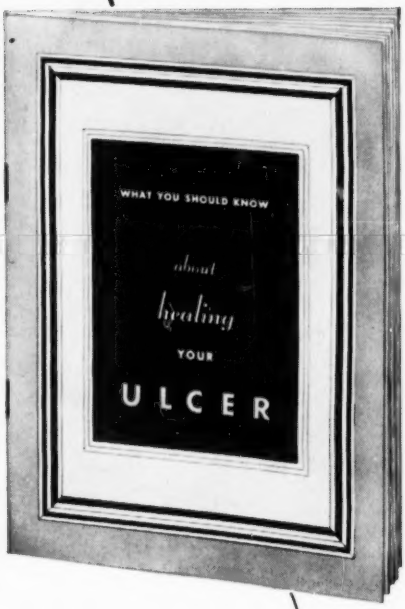
Agoral is emulsified exclusively with refined white mineral oil, purified white phenolphthalein, agar-gel, tragacanth, acacia, egg-albumen and glycerin, by a special process similar to that used for homogenizing milk.

For over 30 years medical men have obtained results with Agoral with a uniformity and precision which are a constant source of satisfaction both to them and to their patients.

William R. Warner, Div. of Warner-Hudnut, Inc., New York 11, N. Y.

Prescribe AGORAL® WARNER

PLEASANT AND GENTLY EFFECTIVE WITHOUT DISTRESS OR LEAKAGE



For your peptic ulcer patients

● This free booklet—prepared by staff members of the New York Hospital, Cornell Medical Center—saves you time and effort in managing your peptic ulcer patients.

The booklet emphasizes the importance of cooperation with you. It tells your patient what he needs to know about his ulcer. To obtain copies, just fill in and mail the coupon below.

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